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ES OF THE HEART

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DISEASES OF THE HEART

NOTE.

On the cover of this book is embossed an outline figure of the chest. If a piece of ordinary note-paper be applied to this, and the point of a black-lead pencil be drawn from side to side over the paper, a "rubbing" will be obtained which will serve as a "chest-chart," and on which the situations of murmurs, the outlines of dulness, &c. can be indicated by coloured marks.

LECTURES

ON THE

PHYSICAL DIAGNOSIS

OF

DISEASES OF THE HEART

BY

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*** A small Edition having been rapidly exhausted, this little Work has been reprinted with improvements in typography and, it is hoped, in arrangement.

October, 1876.

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PREFACE.

THE following lectures were intended for those students who had mastered the rudiments of diagnosis, and who were qualifying themselves for careful observation in the hospital wards. It has been thought that they might prove useful also to practitioners as presenting the essentials for the clinical recognition of Diseases of the Heart, according to the most modern views of cardiac pathology. The work is in no sense encyclopædic. It is intended to be suggestive, not exhaustive, and is founded as far as possible upon personal observation and experience.

The author is conscious of much imperfection of detail, but his aim throughout has been to cultivate habits of close observation. Given a moderate share of logical acumen, and the closest observer will be the best diagnostician.

It will be noticed that no mention is made of the sphygmograph; in the present state of our knowledge, it seems better that this should be studied apart from the ordinary means of physical diagnosis.

29, DUNCAN TERRACE,
March, 1876.

SCHEMA.

I.—Symptomatology.

A. Symptoms referred to the Heart. (*a.*) *Pain* referred directly to the Heart-region in cardiac disease is rare. Organic disease may progress without giving rise to Pain. The special pain of Heart Disease is *Angina Pectoris*, characterised by paroxysmal recurrence, great distress, coldness, arrest of respiration. (*b.*) *Palpitation:* A frequent symptom in Heart Disease, but common in dyspepsia and in emotional conditions. (*c.*) *Intermisson:* Common in Heart Disease, but may be neurosal. (*d.*) *Irregularity:* Generally of grave import, but may also be neurosal.

B. Symptoms referred to the Circulation. (*a.*) *Pulsation:* Excluding emotional causes, suspect Hypertrophy of Heart, and especially *aortic regurgitation.* (*b.*) *Hæmorrhage:* Common in Heart Disease: Not of dangerous import as in Phthisis: *Note* tendency thereto in *mitral stenosis.* (*c.*) *Cyanosis: Vide Inspection.* (*d.*) *Dropsy:* A late and dangerous symptom.

C. Symptoms referred to the Lungs. *Note* that these symptoms are very frequent. (*a.*) Dyspnœa, aggravated by exertion; periodic or persistent. (*b.*) Cough.

D. Symptoms referred to the Brain. (*a.*) Languor and powerlessness. (*b.*) Vertigo and symptoms of disturbance of cerebral circulation. (*c.*) Epilepsy. (*d.*) Chorea. (*e.*) Apoplexy. (*f.*) Paralysis.

Note.—In cases of cardiac hypertrophy coexisting with renal disease, suspect intra-cranial hæmorrhage. In sudden cerebral attacks in patients with valvular disease suspect Embolism.

E. Symptoms referred to the Alimentary Canal. (*a.*) Dyspepsia very common. (*b.*) Hæmorrhoids.

F. Symptoms referred to the Throat. (*a.*) Pain referred to the throat may be a variety of *Angina.* (*b.*) Aphonia occasional in pericarditis. (*c.*) Hoarseness.

G. Symptoms referred to the Kidneys. (1.) Renal disease may be induced by the Heart-affection. (2.) Renal disease may induce hypertrophy of the Heart. (3.) Renal and cardiac disease may be the double effect of one cause.

NOTE.—In cases of Cardiac Disease, always examine the urine, and especially record conditions of albuminuria.

II.—ETIOLOGY.

(*a.*) *Rheumatism* is the most frequent cause of valvular disease of the Heart. *Rheumatic Fever* is in a large number of cases the starting-point, but in other cases the rheumatic symptoms may be very slight and obscure, and sometimes the rheumatic form of endocarditis may occur with no other manifestation of rheumatism.

NOTE.—Examine the condition of the heart in the slight, as well as in the severe forms of Rheumatism.

(*b.*) Scarlet Fever is also a cause of valvular disease, probably because of the rheumatoid phenomena associated with it. (*c.*) The other most common causes of Heart Disease are muscular overstrain, alcoholism, syphilis, tuberculosis, the puerperal state, poisoning by phosphorus, malnutrition, disease contiguous to heart and pericardium, diseases of lung inducing venous engorgement.

NOTE.—A satisfactory examination of every patient, whatever his ailment, cannot be made unless the cardiac conditions are observed and recorded.

III.—PHYSICAL EXAMINATION—INSPECTION.

A. Hue of the surface. (*a.*) Blueness. (1.) *Congenital cyanosis* indicates persistent foramen ovale, or imperfection of inter-ventricular septum, usually combined with obstruction of pulmonary artery. (2.) *Intermittent cyanosis*, common in cardiac dyspnœa.

NOTE.—Chilling of finger-tips with blueness and coldness common in Heart Disease.

(*b.*) Pallor. (1.) When associated with œdema, suspect coexistence of *renal disease.* (2.) With exophthalmos, thyroid enlargement and irritability of Heart, *Graves' Disease.*

(*c.*) Tinge of Jaundice. (1.) In passive congestion of liver in later stages of valvular disease. (2.) With *arcus senilis* in *Fatty Degeneration of Heart.*

B. Cardiac Dyspnœa. (1.) Characterised by gasping air-craving, aggravated by exertion—Orthopnœa; indicative of valvular diseases, or cardiac degenerations.

(2.) Decubitus on right side, with expression of anxiety and apprehension. Suspect *Pericarditis.*

C. Œdema: Commences in lower extremities, serous cavities affected last: Usually depends on *valvular disease* (especially mitral, or conjoined mitral disease and tricuspid insufficiency).

NOTE.—Examine urine, and determine the question of co-existence of renal and cardiac disease.

D. Pulsation (*a.*) In veins of neck indicates *tricuspid insufficiency.* (*b.*) In arteries: Locomotive pulse indicates *aortic regurgitation.* (*c.*) Displacement outwards of visible apex-beat indicates *hypertrophy* or *dilatation of left ventricle.*

NOTE.—Area of visible impulse may be apparently increased at times in nervous palpitation.

(*d.*) Retraction of intercostal spaces coincidently with systole indicates *pericardial adhesions.*

(*e.*) Pulsation of the liver indicates *tricuspid regurgitation.*

IV.—PHYSICAL EXAMINATION—PALPATION.

A. Pulse. (*a.*) Rigid arteries, tortuous, with roughnesses felt in their walls, associated with hard pulse, indicate *Atheroma.*

(*b.*) Slowness of pulse, with very weak impulse, may indicate *Fatty Degeneration.*

(*c.*) Sudden variation of pulse with great acceleration on movement may occur in *Pericarditis.*.

(*d.*) Jerking, collapsing pulse in *aortic regurgitation.*

(*e.*) Feebleness of pulse, and (*f.*) Irregularity and intermission, increased by effort, in *mitral disease* and *cardiac degeneration.*

B. Apex-beat. Displaced. (1.) Upwards by *pericardial effusion.* (2.) Downwards and outwards in *hypertrophy* and *dilatation* of heart.

C. Forcible pulsation felt under false ribs to left of ensiform cartilage indicates *hypertrophy of right ventricle.*

D. Visible pulsation of right auricle indicates *hypertrophy and dilatation of right chambers.*

E. Visible pulsation of left auricle indicates *mitral stenosis.*

NOTE.—That this pulsation may be rendered evident by vibrating levers.

F. Special Cardiac Tactile Phenomena. (*a.*) Friction-fremitus denoting Pericarditis. (*b.*) Thrill. (1.) Occurring with systole over aortic valves denotes aortic stenosis or aneurism; over pulmonary valves, pulmonary stenosis; at apex, mitral regurgitation. (2.) Occurring with diastole at base, denotes aortic regurgitation. (3.) Occurring at apex just before, and terminated by impulse, indicates mitral stenosis.

NOTE.—That decided presystolic thrill at apex is pathognomonic of mitral stenosis, and may occur without murmur.

V.—PHYSICAL EXAMINATION—PERCUSSION.

A. Præcordial area over-resonant or tympanitic—in emphysema of lung, and in the very rare cases of pneumo-pericardium.

B. Præcordial dulness extended. (*a.*) Upwards; outline of dull area being triangular or pyriform with apex at or above third costo-sternal articulation. There is probably *Effusion into pericardium.* (*b.*) Laterally. The heart is enlarged by hypertrophy or dilatation.

I. Lateral dulness extends *left* of normal area. (1.) Area of dulness triangular, apex pointed, *Hypertrophy of Left Ventricle.* (2.) Area of dulness rhomboidal, apex rounded, *Dilatation of Left Ventricle.*

NOTE.—Compare position of apex and outline of left ventricle with evidences of force of apex-beat. If left side enlargement with forcible heaving impulse and long first sound, *Hypertrophy.* If enlargement with feeble impulse and short first sound, *Dilatation.*

II. Lateral dulness extends *right* of normal area. There are *Hypertrophy and Dilatation of Right Auricle and Ventricle.*

NOTE epigastric impulse and signs of tricuspid regurgitation if present.

VI.—Physical Examination—Auscultation.

Section I. The normal heart-sounds are modified in degree.

A. At the *base* of the heart:—

(*a.*) The aortic second sound is intensified. There is *Hypertrophy of the Left Ventricle.*

(*b.*) The pulmonary second sound is intensified. There is heightened tension in the pulmonic circulation.

Note.—A strong pulmonary, associated with a weak aortic second sound, is presumptive evidence of *mitral obstruction or insufficiency.*

B. At the *apex* of the heart:—

(*a.*) The first sound is increased in duration. There is *Hypertrophy of Left Ventricle.*

Note.—When there are signs of Hypertrophy of Left Ventricle without other cardiac signs to account for it, suspect *Chronic Renal Disease.*

(*b.*) The first sound is short, resembling the second sound. There is *feebleness, dilatation or degeneration of left ventricle.*

Note.—Reduplication of heart-sounds occasionally occurs in health; when associated with fever or with grave debility, indicates *myocarditis* or *cardiac degeneration.*

Section II. Abnormal sounds are heard over the heart-region.

A. Generally over the præcordial area.

(*a.*) Friction sound indicates *Acute Pericarditis*—or *Pericardial roughening* the result of a remote pericarditis.

B. Localised in definite relation with the situations of the valves.

(*a.*) First-sound murmur over site of aortic valves (anæmia excluded) indicates *aortic obstruction.*

Note.—The murmur may be localised or propagated in the course of the great arteries.

(*b.*) Second-sound murmur over site of aortic valves indicates *aortic regurgitation.*

Note.—This murmur may be localised, or propagated downwards in the line of the sternum.

(*c.*) Double (first and second sound) murmur (*a* and *b*) denotes combined *aortic obstruction and insufficiency.*

(*d.*) First-sound murmur over site of valves of pulmonary

artery denotes (Anæmia and pressure upon trunk of pulmonary artery excluded) *obstruction of pulmonary artery.*

NOTE.—Pulmonary obstruction is nearly always congenital.

(*e.*) First sound murmur at base of ensiform cartilage indicates *tricuspid regurgitation.*

NOTE.—This is most commonly found as secondary result of mitral disease.

(*f.*) Before-first-sound murmur at base of ensiform cartilage denotes *tricuspid stenosis* or *obstruction.*

(*g.*) First-sound murmur heard over situation of right ventricle may be due to *myocarditis* or *anæmia with cardiac debility.*

NOTE.—These murmurs are not persistent.

(*h.*) First-sound murmur localised in mitral area denotes *mitral regurgitation.*

(*i.*) Before-first-sound murmur localised at apex or just internal thereto denotes *mitral stenosis*—i.e., *obstruction.*

LECTURE I.

INTRODUCTORY.

Symptomatology—Importance of subjective symptoms in regard to treatment: of objective in diagnosis—Pain—Angina pectoris—Cardiac symptoms—Disturbances of circulation—Pulmonary symptoms—Cerebral phenomena—Embolism—Gastric symptoms—Signs referred to the throat—Renal symptoms—Etiology—Rheumatism—Other causes of heart disease.

A FEW words on the threshold of the subject—advice pertinent to investigation of disease in any form, but yet I think rather particularly pertinent to the class of diseases which we shall presently examine. When a suffering patient comes to you for examination, do not permit a diversity of intent between your mind and his. His chief object is to get relief from his aches and pains; yours, perhaps, is to find out—by a process similar to that employed by botanists to discover the name of a newly found flower—the name of the disease which he suffers from. Excuse my saying that you are wrong—your duty is not chiefly to find out what disease he is the subject of, but what his sufferings are, what his deviations from sound health. The plane of unhealth is divided into definite squares, which are called Diseases, and the nomenclature of these is very important, because the mention of one of them conjures up in the medical mind a large picture, and a moving panorama of

phenomena; but your sole object is not to find out into what particular square your patient's aggregate of symptoms will fit. You must investigate his ailments as well as his diseases if you would do him good. I am very far from wishing to be a censor, but I do think that in these days of great and increasing precision in physical diagnosis, there is a real danger of students thinking too lightly of the uttered complaints of patients. It has been said that one objective sign is worth a dozen subjective symptoms, and this, so far as regards diagnosis, has a large amount of truth; but we must recollect that perturbations, which greatly distress our patient, may exist independently of his chief disease, and it is the patient, not the disease, that we have to treat. Let me instance a hypothetical case. A. B. comes to you and, under fire of cross-examination, discloses that he has had rheumatic fever some years ago. You examine his heart and find something wrong with the mitral valve. You dose him, probably help his heart over its presumed difficulties with digitalis, and order the inevitable belladonna plaster to be applied. But your patient at his next visit is not a whit better. It was not his heart which troubled him—that was neither better nor worse than it had been for many years past, and was likely to be for many years to come; but he suffered from a *trigeminal neuralgia*, and would have showered blessings upon you had you relieved him.

The moral I would point, then, is this: Always commence your interrogatory of a patient by asking what he complains of, what are his troubles and distresses, and if you record his case, write on the first line, *complaint*, and give his expressions, as nearly

as possible, with the necessary abbreviations, in his own words.

Now we come more closely to our subject. The comprehensive view of what disturbs the equanimity of a patient, with a view to the alleviation of his troubles is one thing, the particular determination of the physical significance of his ailments, and the precise diagnosis of his diseases is another. For this latter purpose the details of pains and aches, though all due weight must be given to them, are very deceptive. How very common it is for a patient to fancy that he has disease of the heart because he experiences discomfort or pain in the heart-region, and to insist on being "sounded," so that his doubts may be set at rest. How rarely do we find that he really has organic heart disease? Oftentimes the patient goes away apparently with a look of disappointment that his fears are not confirmed, and with a doubt probably of our skill, because his aches are so precise.

In looking over the notes of a hundred cases of the coarse and decided forms of disease of the heart which have been under my own immediate care, I find that in just half the number, there was no complaint whatever of any pain referred to any part of the chest. Seventeen referred the pain generally to the front of the chest, fifteen to the back of the chest, and especially between the scapulæ. Twelve referred the pain to the epigastrium; eleven suffered pain on the left side of the chest, whilst two referred their sufferings to the right side. Those who localised their sufferings to the exact area of the heart were but eight; and of these two complained of it only on exertion; one referred it to the base of

the heart; one described it as a sense of extreme soreness at the apex; and in one, it partook of the character of neuralgia of the left breast.

So, as far as the evidence derived from the above cases—which are, I believe, fair specimens of those presenting themselves at the public or the private consulting room—teaches, only eight per cent., or less than one in a dozen, complain of pain directly referred to the situation of the organ which is diseased. This, though strange to the non-medical mind, can scarcely be surprising to us when we consider that the heart possesses very little common sensation, and that its structure can be punctured, torn, or lacerated, without the direct infliction of pain. When we consider the vast number of patients who present themselves with symptoms referred to the region of the heart, but really caused by dyspepsia or pleurodynia, and then take into account the very small proportion of real undoubted heart cases presenting signs of local pain, we must estimate at a very low figure the value, in a diagnostic point of view, of *pain* in relation to heart disease.

Looking on diseases of the heart in general from the point of view of symptomatology, we find that their origin and progress are oftentimes very insidious and obscure. Even the most pronounced and dangerous forms of the disease may, in some cases, go through their stages without betraying themselves by marked symptoms of distress. This fact was strongly impressed upon my mind by a case which was under my care at the North Eastern Hospital for Children. A little girl, who had been treated previously for a valvular affection, was brought to me because the mother thought she seemed a little languid, though

the child complained of no distress. On examination, I found that there was effusion into the pericardium. She was admitted, and went through all the stages of pericarditis, with extreme distension of the pericardium, one of the most pronounced instances of pericardial friction that I have ever heard, and an accompanying endocardial change, and yet throughout her whole illness the child complained neither of pain nor distress, and it was impossible to keep her in bed.

There is one form of pain in heart disease, however, which is of terrible significance—I mean Angina Pectoris. This is the pain *sui generis* of cardiac origin, and if you have once seen a pronounced example of it, you will never forget it. The patient suddenly sits up in his bed, and with a cry of horror indicates his sense of pain at the præcordium. This pain is of great intensity, but is of a cold and sickening character; the chest is fixed, the breathing not quickened, and the hand, placed over the epigastrium, finds that the heart's action is slowed and enfeebled. The face wears a look of horror, the hue is pale or slightly leaden, and a cold sweat breaks out upon the forehead. Worse than the pain is the feeling of fearful sinking and depression; the poor patient gasps, "I shall die!" and, sometimes, as in a case which it was once my lot to witness, his short, but concentrated sufferings in a few minutes end in death. The kind of disease which such symptoms especially indicate is *degeneration* of the heart. It is not easy to trace the pathological causation, but it is evidently a neurosis of the sympathetic. All pain, in my opinion, can be referred to a condition of depressed vitality, of impaired nutrition, of certain nerve-centres. In these cases there is, through faulty blood-distribution, an

impaired nutrition of certain portions of the spinal origin of the sympathetic fibrils distributed to the heart-ganglia. As to the immediate method of influence, whether there be induced a vaso-motor spasm or no, is a question too extensive to be entered upon here. From such a typical case of high diagnostic and prognostic import, there are many gradations of intensity until, in some cases it is difficult to differentiate the symptoms from those of dyspepsia or hysteria. In some cases the pain is absent though the other symptoms are present; such is the "angina sine dolore" of Gairdner. The points to be remembered are:—(1) The attacks are paroxysmal with long or short intervals. (2) There is always a sense of coldness experienced, and frequently a cold sweat. (3) The heart's action is *not* increased, and (4) The chest is fixed and the breathing slow. Out of the hundred cases I have mentioned thirteen presented anginoid symptoms in greater or less degree, and of those nine were accompanied by pain. There may or may not be valvular lesions, but probably degeneration of the muscular tissue of the heart is necessary for the induction of the symptoms.

You have seen, then, that a large number of heart cases occur without the manifestation of any pain—that the special pain of heart disease, though of great diagnostic importance, is of comparatively rare occurrence. You may be led to inquire what are the other symptoms that lead up to the suspicion of heart disease; and, although we cannot enter at all deeply into symptomatology, we may endeavour to give a brief answer to this question.

In this review of symptoms, in order to give a rough idea of the relative frequency, I shall place

between brackets the number of instances in which each has been observed in the hundred cases which we have taken as typical. The first class of symptoms (A), excluding pain which we have already considered, includes palpitation and disturbances of the muscular structure of the heart. *Palpitation* is far from being a pathognomonic sign of heart disease. Its first cause resides often in the nervous system. It is not by any means a sign of heightened functional activity, but is rather the "spurt" of overtaxed and wearied muscle. Though occurring very often in conditions in which the heart is not structurally diseased, palpitation is a frequent sign and source of trouble in organic heart affections (28). It is specially called forth on exertion, often of the slightest degree, and it occasions much distress; in some cases, especially where the aortic valves cannot close perfectly, the patients complain that the heart beats like a hammer. Another sign of imperfect action of the muscle of the heart is *Intermission*. After a number of pulsations at regular intervals, the heart waits over the whole period necessary for a contraction, and then resumes, to wait again after another interval. Intermittency, like palpitation, may be no sign of structural heart disease; it is in many cases due to an incoördination of nervous actions, and is to be ascribed, not to cardiac, but to cerebro-spinal causes. When it does occur, however, in ascertained organic disease, it is of serious import. It probably means that the contraction of the auricles is at certain times so imperfect that they do not fill the ventricles. The necessary stimulus to the ventricular contraction is a sufficient repletion of the cavities. Hence the ventricles wait until the auricles supply

them with enough blood. A third trouble of heart-muscle is *Irregularity*. This, also, is due to a want of action in accord of the layers of muscular fibre of which the heart consists. The heart does not wait for a whole beat as in intermission, but alters its rhythm irregularly. Sometimes the left and right sides do not contract, or rather do not complete their contraction, at one and the same moment; then the action is *reduplicate*. Sometimes there is so much disturbance that the periods of action and rest cannot be discriminated; the beats are *tumultuous*. Irregularity may, like the other disturbances of heart-muscle, exist independently of organic disease; but when it reaches a high degree, it is one of the strongest evidences of such disease. Patients sometimes describe the irregular action as like the fluttering of birds. One complained to me of a fluttering in his heart "like two pigeons." Such signs are of dangerous import.

The symptoms, however, may be referred, not to the heart itself, but to the next portion of the system of blood-distribution—to the arteries. Patients complain of (B) *Pulsation*. This, we shall see, is very pronounced in the general arterial system when the aortic valves are incompetent to close. It was complained of in my patients as a symptom of distress (3) in the ear whilst lying on the pillow, in the right temple, in the occipital region. Pulsation is, of course, a characteristic sign of aneurism, but this condition we do not intend to discuss. Another arterial symptom in connexion with heart disease, but rare and of no considerable diagonistic importance, is *Flushing*. A very important consequence is (C) *Hæmorrhage* (22). This may occur from the

lungs (12), when it must be remembered that it has not the dangerous significance of the hæmoptysis of pulmonary phthisis. Considerable quantities of pure blood can be expectorated with the result of relieving the venous engorgement of the lung, which is the result of some forms of heart disease. Of worse omen, in my opinion, is the frequent voiding of blood-stained sputa; this occurs usually when the right side of the heart is dilated, and is one of the late consequences of valvular disease. Bleeding from the nose (5) is far from uncommon in heart disease; it may occur also from the stomach (3), or from the uterus (metrorrhagia) (2). In all these cases the hæmorrhage may occur from the direct rupture of capillaries by the shock of an unduly contracting left ventricle, but much more commonly it occurs from superinduced *venous* congestion. Occasionally the hæmorrhage is not manifest outwardly, but occurs in the interior of organs.

Other signs are referable to plethora of the venous system. The veins may sometimes be seen to be obviously distended, and in some cases, as we shall describe, the large veins pulsate. Patients with certain malformations of the heart exhibit a blueness of surface (D), *Cyanosis*, from the distribution of venous blood by the arterial channels. A like blueness obtains intermittingly, on account of venous congestion, in the paroxysms of dyspnœa from which patients with heart disease occasionally suffer. A consequence of habitual venous plethora may be (E) *Dropsy*, of which it is well known that cardiac disease is one of the great inducing causes.

Next in order to the obvious disturbances of the heart itself and the channels of blood-distribution, we

come to consider the symptoms of disturbance of the functions of the *lungs* in heart diseases. These symptoms are far more frequent than those referred to the heart itself. Nearly half the cases (45) complain of difficulty of breathing. Some (8) are obliged to sit upright (orthopnœa) in order to breathe. A very large proportion of patients with heart disease suffer from cough (45).

A great characteristic of the dyspnœa of heart disease is, that it is produced or aggravated by slight exertion. The heart may fairly accommodate itself to conditions of rest, but let exertion call upon it for increased action and it manifests its distress by the imperfect pulmonary circulation, and the consequent dyspnœa. Or the dyspnœa may be periodic, and not induced by voluntary effort. Such attacks are called cardiac asthma. The induced conditions may, however, be not temporary, but chronic. Persistently defective heart's action induces persistently defective pulmonary circulation. The blood tends to stagnate in the lungs. Chronic bronchitis, and, subsequently, emphysema follow, and the trouble may be augmented by œdema of the lungs.

Next we will proceed to notice the *Cerebral* troubles which occur in heart disease. These are very common. The patients complain of languor and extreme weakness (25). Often the muscular weakness is referred especially to the arms; the patients cannot lift weights as they have been accustomed, and the muscles feel powerless. There are attacks of giddiness (vertigo) (8), or they are subject to faintings (syncope) (6). There may be an undefined nervousness (7), with dread of a fit or some calamity, and lowness of spirits. Headache (5) is sometimes met

with, but is not one of the commonest symptoms. Trembling of muscles and the irregular jactitations of chorea (3) are very important to notice. There may be fits epileptiform (2), or epileptic (2), or various forms of paralysis may be found. Lastly, there may be impaired memory and intellectual disturbance of various kinds. In all cases of heart diseases which present cerebral symptoms, the fundus of the eye should be examined by the ophthalmoscope.

The cerebral phenomena observed in heart disease may, for the most part, be divided into three classes. The first embraces those due to the *chronic* disturbance of balance between the arterial and venous systems which is the result of imperfection of the driving power in the great engine of the circulation. The brain may suffer from deficient supply of arterial blood, or from excess of venous blood, or from these causes variously combined. Arising from these conditions there may be increase of the fluids effused within the intra-cranial cavities, and degenerations of brain tissue, owing to the impaired nutrition.

The second class of cerebral phenomena occurring in heart disease includes those due to intra-cranial hæmorrhage. You must remember that apoplexy is to be feared in cases of hypertrophy of the left ventricle of the heart, the strong muscular contraction distending the arterioles and capillaries to the point of rupture. It is much more to be feared, however, when there is heart hypertrophy and kidney disease combined. In such cases there is not only excess of driving power, but the arterioles have suffered change—they have become brittle and prone to rupture. Out of twenty-two cases of apoplexy, Kirkes found thirteen accompanied by hypertrophy of left

ventricle, and fourteen accompanied by renal disease; and Eulenberg, in six cases of apoplexy, found five with contracted kidney and heart hypertrophy.

The third class includes the interesting phenomena now known to be due to the sudden blocking of a cerebral artery by a morsel of coagulum detached from a diseased portion of endocardium, and swept onwards in the blood current until it happens to be arrested in an arterial channel which is too large to pass through. The straightest course which such a plug can pursue is from aorta to middle cerebral artery of the left or the right side, and the symptoms produced are hemiplegia, with coma, or aphasia. Sometimes, however, the effects are more chronic, the plugging of the cerebral vessel, and hence the cutting off of nutrient supply, inducing softening of that portion of the brain supplied by the vessel.

Instead of a large plug of this sort we have reason to believe that small ones which are arrested in the cerebral arterioles occasionally occur, and may account for symptoms for which no cause has been discovered. Thus in chorea, which has a notable connexion with disease of the heart, it has been suggested by Dr. Hughlings Jackson that there are embolisms of the arterioles of the corpus striatum and the adjoining convolutions.

Embola may be carried in the blood current to other parts than the brain; the spleen, liver, and kidney can be thus affected. Capillary embolism of the kidney is probably not very uncommon. It is suggestive that in two or three cases I have noted, in which chorea occurred in rheumatic endocarditis, frequent micturition was a symptom complained of.

We turn now to another set of symptoms in heart

affections, those of the *stomach.* Pain referable thereto we have already noticed. A large proportion of patients with heart disease complain of some of the symptoms of indigestion—gastric catarrh is common; nausea, vomiting, pyrosis, and flatulence are frequent, and occur in a circle, the heart trouble occasioning them, and the symptoms reacting to cause palpitation and heart distress. The other abdominal viscera also partake of the venous plethora induced in heart disease. Hæmorrhoids are frequently met with.

Another set of symptoms in diseases of the heart comprises those referred to the *throat.* This subject presents, in my opinion, a wide and very promising field for observation. Pain beginning at the throat is referred to by some as a very dangerous sign in these affections. There are many instances on record in which a patient has grasped at his throat, evincing signs of acute pain, and has shortly afterwards expired. The sudden throat pains in heart disease are, I believe, for the most part, varieties of angina, and may be discriminated by the rules I have given. Other forms of pain of less laryngeal character and of less intensity are, however, met with, and these are usually accompanied by flatulence and accompanied by dyspepsia. The "rising in the throat," unaccompanied by pain, is usually either dyspeptic or hysteric. Next to the throat-angina, the symptoms of greatest interest and importance are loss of voice (1) and hoarseness (4). The case of aphonia which I have noted occurred during the progress of pericarditis—to what was it due? A case which suggests an answer to this question is given by Dr. Morell Mackenzie in his book on "Hoarseness and Loss of Voice." In this instance pericardial effusion was accompanied by

aphonia due to paralysis of the adductors of the vocal cords. After the cessation of the pericarditis the mobility of the affected laryngeal muscles returned. The pathological process whereby such paralysis is brought about is yet undiscovered. Unfortunately in my case the laryngoscope was not employed; it teaches, however, that it should be always used, if possible, under like conditions in the future. We may next ask, to what may the minor affection—hoarseness—be due in heart disease? We find that hoarseness occurs as a symptom of aortic aneurism, the disposing cause in such case being pressure of the aneurismal sac on the left recurrent nerve, inducing paralysis of certain of the laryngeal muscles. I have not found, as far as my own experience goes, that hoarseness is prone to occur in aortic valvular diseases. The cardiac conditions accompanying hoarseness I have found to be disease of the mitral valve, with which broncho-pneumonia, or some other form of pulmonary mischief, co-exists. Unilateral paralysis of the intrinsic muscles of the larynx is frequently met with in local disease of the pulmonary texture.

Disease of the *kidneys* may stand as to disease of the heart in a threefold relation. The latter may be cause, consequence, or concomitant. The renal disease may be (*a*) directly caused by the heart-imperfection. The kidneys, like the other viscera, suffer venous engorgement, and, if this be long continued, a low form of inflammation may occur in them. In the earlier stages such a state of things is indicated by albuminuria, in the later by the detection of kidney tube-casts in the urine by the microscope. But (*b*) the heart disease may be primarily caused by the renal disease. When, owing to structural disease.

the kidneys are unable to excrete from the blood the urinary solids, the natural consequence is the retention in the circulation of effete material. It is supposed that this material perfunctorily retained so irritates the arterioles, or the vaso-motor centre which governs them, as to cause them to contract. Such contraction (it seems to me that the term "spasm," which has been used in respect of this effect, is misapplied) being long kept up, the result is the same as occurs in overtaxed muscle-tissue generally—viz., hypertrophy; so there is induced a peripheral obstacle to the onward current of blood in the arteries. The heart struggles against such an obstacle, and its efforts produce hypertrophy and dilatation of the left ventricle. Or it may be (*c*) that the heart disease and the kidney disease are both effects of one cause. The form of kidney disease which usually accompanies cardiac hypertrophy is what is called "contracted kidney;" this is for the most part associated with some gouty affection. We know that in gouty disease the blood is impure, and it is supposed that such condition gives rise to a disease of arteries and capillaries generally throughout the system leading to thickening of their walls, but not necessarily of the muscular part thereof. The heart hypertrophies because it struggles against the obstacle, not of arterioles actively contracting, but of tubes unduly rigid. The kidney is diseased because its arteries are involved in the general disease. Concerning the conditions *b* and *c* we have to use the term "may be," for the medical mind is not made up as to their pathology; certain it is, however, that renal disease is especially associated with cardiac hypertrophy. The rules you can deduce from a con-

sideration of the whole subject are: 1. In cases of cardiac disease carefully examine the condition of the urine. 2. When you find renal and cardiac disease co-existing, weigh carefully the facts of the previous history, and endeavour to find out which pathological condition preceded the other.

I turn now to another branch of the subject. I have said that the symptomatology of heart disease is often obscure. We find that its etiology is often obscure likewise.

There are two errors, in my opinion, into which many are prone to fall. The first is that valvular disease of the heart is rarely found except as a consequence of rheumatic fever; the second that there is danger of heart-complications in the severer forms of rheumatism only. Let us turn to the records of actual cases. Taking seventy-seven of the hundred cases before cited, in which the early histories are sufficiently precise, I find that thirty-four occurred in those who had suffered one or more attacks of undoubted rheumatic fever; but in thirteen there had been rheumatic pains only, not sufficient to keep the patients to their homes; and in fifteen there was no history of any rheumatic affection whatever, and only, if any symptoms at all, those of a lightly-regarded indigestion. Rheumatic gout had been suffered by two patients, scarlet fever by three, and typhoid, or "low" fever, by four. In six cases the evidence pointed to the conclusion that the disease was congenital. You will conclude, therefore, that, though it is (1) pre-eminently necessary that you should carefully examine the condition of the heart in any patient who is suffering from, or who has suffered from rheumatic fever, it is important to do

so also in (2) those who have been subject to slight forms of rheumatic pain, and that (3) there is a large remnant requiring careful exploration whose diseases are not to be traced to any obviously rheumatic condition.

Excluding, then, those I have mentioned, what are we to look for as the most common causes inducing heart disease? I will briefly enumerate some of them: Over-exertion and muscular strain—alcoholic indulgence — syphilis — tuberculosis — the puerperal state—poisoning by phosphorus—imperfect and improper nutrition—disease which involves the structures contiguous to the heart and pericardium—disease which induces venous engorgement of the lung, and hence distension, dilatation, and hypertrophy of the right side of the heart.

You may, from a general consideration of this introductory chapter, obtain in some degree an answer to the question which you will probably propound:—Under what circumstances of symptoms and previous history is it necessary for me to make a physical examination of the heart-region? There is one aphorism which I would impress on you, however, which covers the whole ground. It is this, *that you have never made a complete examination of any patient, whatever be his ailment, unless you have estimated, as far as possible, the condition of his heart.*

We shall now consider the mode of doing this. We pursue the investigation through our senses of sight, touch, and hearing. We do not grope for one sign which, when found, shall be conclusive to us; but after we have obtained all the evidence presented to our senses, our logical faculty must discriminate and lead us to the truth. We work by no single method, but by a combination of methods and modes of thought.

LECTURE II.

INSPECTION.

Cyanosis temporary and permanent—Pathological causation—Chilling of finger-tips — Clubbing — Anæmia — Graves' Disease—Sub-icterus—Arcus senilis—Cardiac dyspnœa—Orthopnœa—Decubitus in pericarditis—Œdema—Venous turgescence—Venous pulsation—Visible arterial pulsation—Locomotive pulse—Apex-beat—Area of visible cardiac impulse.

You may take it as an aphorism that you can never make a satisfactory examination in suspected heart disease, unless your patient be stripped to the waist. Very valuable evidence is afforded by inspection. The first point you will probably note is the general *hue* of the surface.

We will suppose that there is (A) a marked blueness of the surface. You will elicit whether this is temporary, sometimes passing away altogether, or permanent, varying perhaps in intensity, but never quite disappearing. The temporary blueness will be associated, probably, with attacks of cardiac asthma, of which dyspnœa is the great feature. The permanent blueness may also depend on the same cause as the temporary—viz., undue fulness of the venous system. In such case you will find respiratory trouble—bronchitis or emphysema or both accompanying it—and it affords strong presumptive evidence of dilatation of the right cavities of the heart.

There is a form of blueness dependent on malformation of the heart so special that it constitutes the chief sign of, and gives a name to, the affection. Such is blue disease, morbus cæruleus, or CYANOSIS. Here you find a deep blue discoloration, in some cases approaching a black, involving all the surface, but especially manifest in the lips and the mucous membrane of the mouth. The colour is persistent, but is deepened when breathing and the heart's action are quickened, or when cough comes on. You will scarcely find any difficulty in recognising this condition—the hue is so characteristic; it is more pronounced and more general than that which obtains in ordinary venous congestion. Moreover you will elicit perhaps that the affection dated from birth. If so, the evidence is nearly conclusive, but, if not, the diagnosis is by no means set aside, for the discoloration may not be obvious until periods remote from birth. Most probably, however, your patient will be an infant or young child. Nearly half the cases of this affection die before they are a year old; two-thirds before they are two years old; and, though a few instances are recorded in which adult life has been attained, they are very rare.

We may now inquire, what is the pathological significance of the phenomenon? As a matter of fact, in a case of cyanosis the chances are rather more than ten to one that there is an abnormal communication between the right and left cavities of the heart, either between the auricles, owing to patency of the foramen ovale or between the ventricles, owing to imperfection of the inter-ventricular septum. Furthermore, the chances are about six to one that the pulmonary artery is obstructed. The cause of the

blueness, according to John Hunter, was held to be the admixture of venous and arterial blood in the circulation. This explanation seemed simple enough. Owing to the structural defect in the heart, the dark-coloured venous blood mixed with the arterial, and the resulting darkened compound was propelled through the systemic arteries. This theory has been opposed in modern days, but it appears to me that its opponents try to prove too much. At any rate they hold that the explanation given above is *not* the true one of the cyanosis. According to them, cyanosis is due to congestion of the venous system, and this congestion is the result of obstruction of the pulmonary artery, or of some other malformation which induces an obstacle to the return of blood from the systemic veins. Premising that, in my opinion, the truth lies between these two theories, we will examine each of them.

The theory that the blueness results from direct mixture of venous with arterial blood at first sight appears very plausible. Out of one hundred and ninety-five cases recorded by Stillé and Peacock, one hundred and seventy-eight presented abnormal communication between the right and left sides of the heart; admixture of venous and arterial blood therefore is possible and likely, and often inevitable. The hue of the patients is just that of those in whom venous blood is circulating: you see it in cases of impending suffocation; you may have many opportunities of witnessing it during the administration of nitrous oxide gas, when the blood is rendered of the venous colour by the excess of carbonic acid which it cannot get rid of. What are, then, the objections to the theory? The first objection is

afforded by the anatomical exceptions. Seventeen of the cases recorded presented no possibility of the arterial and venous currents abnormally commingling. This is sufficient to prove that the cyanotic tint cannot be *always* due to the arterio-venous anomaly; but this fact we knew before. The second objection is that we can have communication between right and left heart without the appearance of cyanosis. This does not seem to me a fatal objection. The question is one of degree; the tint of the arterial blood may mask that of the intermixed venous blood, or *vice versâ*. Nay, more, a patient having this anomaly may present the peculiar coloration at one time and not at another. You may understand this by observing the differences in hue of a woman in health and in a state of anæmia and chlorosis. Let the blood corpuscles be numerous and healthy, and they would mask the coloration due to venous admixture, but let them be by any cause diminished in number or in colour, and the dark tint would declare itself.

Now, let us turn to the other theory—that the coloration is alone due to venous congestion. The anatomical argument weighing with those who uphold this theory is that obstruction of the pulmonary artery is an important and frequently-observed condition in cyanosis even when there is communication between right and left heart. But by their own data it is shown that this sign is less constant than the arterio-venous communication, the chances of the first condition being about six to one, the chances of the second more than ten to one. Moreover, it is an ascertained fact that there may be great obstruction of the pulmonary artery without cyanosis. A case

under my care at the North-Eastern Hospital for Children showed this most positively. A little girl, aged eight years and a half, presented signs of extreme anæmia; there was no blueness, but great pallor; all over the heart region was heard an extremely loud murmur with the first sound; it was loudest at the base of the heart. If our patient had been an adult, we should have had very strong reasons for concluding that the murmur was due to anæmia. At the autopsy we found that there was obstruction by narrowing of the pulmonary artery. This was the only morbid condition to account for the murmur. It is evident, then, that there can be great obstruction of the pulmonary artery without cyanosis. It is, however, undoubtedly true that in some cases of blue disease, though these are infrequent, there has been found no communication between right and left heart, but only an obstruction upon the venous side which has given rise to a general congestion of the veins of the system.

From these considerations I think we are justified in arriving at these conclusions—first, that in a large number of instances of congenital cyanosis there is abnormal communication between right and left heart, and it is scarcely reasonable to doubt that the circulation of venous blood with the arterial tends to produce the peculiar blue coloration; secondly, that in some cases the blueness is produced only by undue fulness of the superficial veins. We know that such blueness can be thus produced, because we see it during attacks of dyspnœa, which spring from many causes, and we are familiar with it when severe cold affects the surface of the body. In this latter condition the cold causes contraction of the arterioles,

and these force the blood onwards into the capillaries and venous radicles.

Having recognised your case as one of congenital cyanosis, you are by no means to stop here, but to examine by all the methods which will be detailed hereafter, in order to determine as far as possible in what the malformation consists.

There is one point in cases of venous obstruction from any cause which may be classed under minutiæ, but which is of considerable diagnostic importance—the condition of the finger-ends. Notice if the finger-nails are blue in colour. If they are persistently blue, you may conclude that there is persistent fulness of the venous system. If they are occasionally blue you will find the blueness coincident with attacks of dyspnœa. Notice also the temperature of the finger-ends so far as it is manifest to touch. Coldness means great defect of circulating power. Blueness and coldness together constitute a measure of the danger of attacks of cardiac asthma, and when these signs are persistent in cardiac disease they show that the end is not far off. Notice next the *shape* of the finger-ends. When the return of blood from the veins to the right heart is obstructed to a considerable degree and for a protracted period, the finger-ends become thickened at their extremity, or *clubbed*. Dr. Dobell has called attention to a very important practical point: the relation of the clubbing of the finger-ends to the shape of the nails. Clubbing of the finger-ends is well known to occur in phthisis as well as in heart disease. The conditions are to be thus discriminated:—When there is symmetrical clubbing of the finger-ends, and the nails are of the normal shape, the chances are in favour of heart

disease; when the clubbing is attended with curvature of the nails over the ball formed by the finger-tips, the chances are in favour of phthisis. The reason, probably, is that in the one case the adipose tissue exists in its normal quantity, and in the latter case it has wasted, just as in phthisis all the adipose structures waste.

We will now turn to another branch, and suppose that our patient does not present blueness but (B) pallor. The skin, the mucous membrane of the lips, the gums, and the conjunctival surface of the eyelids are pale; the sclerotic portion of the eyeball is of a pearly white. The patient, you say, is bloodless—anæmic. Anæmia may simulate heart disease, may be produced by heart disease, may aggravate heart disease. The method of differential diagnosis between the heart disturbances induced by anæmia and by organic heart disease respectively we shall hereafter consider, but there are two conditions of anæmia which we may with advantage notice here.

The first is the condition of pallor associated with renal disease. We have seen in our introductory lecture that there is an especial relation subsisting between cardiac and renal disease. It is most important to discover such co-existence. If, in addition to pallor, you notice a "puffing" beneath the eyelids, or other signs of œdema about the face, you may suspect renal complication, and prepare (1) to examine the urine, (2) to use the ophthalmoscope. You will determine the specific gravity of the urine, ascertain whether it is albuminous and examine its sediment by the microscope. You will examine the fundus of the eye to ascertain whether certain changes which are known to co-exist with albuminuria are present. The

red field of the fundus oculi presents in albuminuric retinitis irregular, or star-shaped, white patches, with, sometimes, dots of apoplectic extravasation.

The next condition usually, if not always, associated with anæmia, which we shall consider, is known as Graves' or Basedow's disease. This peculiar affection, by a strange misplacement of an adjective, has been called exophthalmic goitre. It is characterized by a triple sign; (1) prominence of the eyeballs; (2) enlargement of the thyroid body; (3) irritable action of the heart. The prominence of the eyeballs (exophthalmos or proptosis) is readily distinguished. The globe seems to be pushed forward, and the effects may vary in degree from an appearance, at first sight, of merely an unusual size of the eyeball to a most unnatural protrusion—to such an extent that the eyelids cannot cover it, and the cornea becomes dimmed by inflammatory changes. The thyroid enlargement varies much in degree. In the cases I have seen, it has not been symmetrical; of three cases the enlargement was chiefly of the right lobe in two. The swelling is elastic to the touch, and pulsation of the arteries is readily felt. The beating of the heart is visible over a wide area; the action is very rapid, excited by the least emotional provocation, and often exceeding 120 per minute. This affection does not rank as a heart disease, but it may lead to hypertrophy and dilatation. It is really an affection of the sympathetic nerve, whereby its "vis nervosa" is impaired. Paralysis of certain of its cardiac ganglia and fibrils induces the palpitation. Arrest of function of the vaso-motor branches distributed to the thyroid arteries induces the hyperæmia and enlargement of the thyroid body, as can be synthetically shown in

animals by division of these fibrils; and the prominence of the eyeballs is caused by a like influence, producing a dilatation of arteries and veins at the back of the orbit.*

We will now suppose that our patient presents neither blueness nor pallor, but (C) *a yellowish tinge* of the surface. Deep jaundice is not frequent in heart disease, but a slightly icteric hue of the face, with a more deeply-tinged conjunctiva where it covers the sclerotic, is not uncommon in the later stages of valvular disease when passive congestion of the liver is one of the troubles. When you find your patient past the prime of life, the victim perhaps of alcoholism, with a generally dusky yellowness of the skin, but without jaundice, the surface of the body somewhat greasy to the touch, the muscles felt to be flabby and found to be weak, patches of dilated capillaries upon the face, and venous turgidity of the conjunctiva—when, moreover, you notice that breathlessness or faintness is produced on exertion—you may fear fatty degeneration of the heart. For the diagnosis of this affection one sign has been adduced especially, and has perhaps sometimes been too strongly relied upon—the presence of an *arcus senilis* in the cornea. This requires careful scrutiny, for there are arcus and arcus. Near the junction of cornea and sclerotic, where the former should be clear

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* For a good *résumé* of the yet known facts concerning this affection, the reader is referred to an abstract of a paper by Mr. H. T. Shapley, Resident Medical Officer at the London Hospital, in *The Students' Journal* for June 20th, 1874, p. 196.

For details vide "Diseases of the Heart and Aorta," by Dr. Hayden, p. 1050 et seq. Dublin: Fannin & Co. London: Churchill, 1875.

and transparent, you may see a circle, a semicircle, or a crescent of opaque whiteness. If this be well defined, and the rest of the cornea bright and translucent, it is, probably, no indication of internal degenerative change. The corneal opacity is probably due to calcareous infiltration. But if the ring be ill-defined, rather yellowish than white, the rest of the cornea being slightly cloudy, you may consider that the chances of cardiac degeneration are formidable.

Having learnt the lessons to be deduced from the complexion and hue of our patient, we will next consider the points to be gained from observing his mode of breathing and the postures which he assumes. In this section we will consider first cardiac dyspnœa. As I have said in the introductory lecture, this symptom is especially provoked by exertion. Thus it differs from dyspnœa of pulmonary origin. You may find your patient breathe easily enough if he has been still for a short time previously to your examination, but if you call upon him to walk briskly for a few minutes, or especially if he ascend a few stairs, breathlessness comes on. This dyspnœa is peculiar—there is no real obstruction either to inspiration or expiration, but the patient gasps restlessly, there is an instinctive craving for more air to oxygenate the sluggish blood in the lung; there is *air-hunger* as the Germans expressively term it. Such is the dyspnœa of the earlier stages of valvular imperfection; but in the later stages the symptoms may be far more distressing. The sign which especially distinguishes the dyspnœa of the later stages of cardiac disease is *orthopnœa*—the patient cannot lie down; perhaps can scarcely recline from the perfectly upright position: the whole mental energies seem bent upon the

one task of getting air into the chest. For a description of the most extreme condition of cardiac dyspnœa, I will quote the graphic words of Hope. The patient, "incapable of lying down, is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forwards and the elbows or forearms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa; sometimes, however, extending the arms against the bed on either side to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanding and starting, eyebrows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts around a hurried distracted look of horror, of anguish, and of supplication; now imploring in plaintive moans or quick, broken accents and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine, and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings." When the conditions of cardiac dyspnœa have long continued you may find a new condition established, more merciful, but of even more fatal augury—carbonic acid poisoning. The blood is poisoned by the carbonic acid, of which the respiratory effect is powerless to disembarrass it. The patient is in a constant state of drowsiness, with difficulty aroused, but waking in distress every now and then when the instinctive craving for more air asserts itself. In the end coma supervenes.

Such are the modes of comportment in chronic diseases of the heart wherein dyspnœa is a feature.

In some cases of *acute pericarditis* you observe no respiratory trouble. Your patient usually lies upon his back, sometimes desiring that his head and shoulders should be raised; if he turns, he prefers to lie upon his right side, because when on his left side, there is not only more direct pressure upon the pericardium, but the liver tends by its weight in this position to press upon the heart. It is usual in pericarditis for a patient to be very unwilling to change his position; syncope may be easily induced by rough movements, and it is to be remembered that such syncope may be fatal. The expression of countenance in pericarditis is often that of anxiety and apprehension, and wandering of the mind is common. Dyspnœa, such as we have described in relation with chronic heart disease, may occur in pericarditis, but you must not look for it as a common symptom. It occurs when the fluid effused into the pericardial sac mechanically obstructs the action of the heart, or in cases where a less amount of effusion weakens an already weak heart.

The "reason why" of all the forms of cardiac dyspnœa is not very easy to trace. In some cases the desire for the upright position is probably due to the relief from the pressure that the diaphragm, impelled by the abdominal viscera, occasions upon the right ventricle which such position induces. In others, where there is fluid in the pericardium, this relief is enhanced by the gravitation of this fluid to the most dependent parts of the pericardial sac, and thus the easing of the heart-muscle from pressure.

Having observed the general hue, posture, and mode of breathing, you will notice whether there are dropsical swellings of the surface of the body, pitting

with the pressure of the finger, or whether there is evidence of fluid in the abdomen. Particularly inquire in what situation the œdema first manifested itself. It is an almost invariable rule that the dropsy, which depends upon cardiac disease, commences at the feet and gradually extends over the lower extremities. Thus it is distinguished from dropsy dependent upon renal disease, which commences in the face. Let me urge you, however, to receive the evidence of patients, when interrogated as to the locality in which the swelling first appeared, with considerable caution. The swelling of the face may have been transient, and even entirely overlooked. I must reiterate the rule which I have given you before—always examine the urine for albumen. If you find albumen present, you must still proceed with your examination of the heart, for a cardiac complication may exist with the renal disease. If you do not find it, you must hesitate before coming to the conclusion that the dropsy is cardiac, for it may be due simply to debility and anæmia. When the patient presents the concurrence of dyspnœa on exertion with a swelling of the feet which pits upon pressure, there is a strong presumption of cardiac disease. Cardiac dropsy proceeds upwards from the more depending parts, involving, after the legs and thighs, the scrotum and the general areolar tissue of the body (anasarca). The serous cavities, the peritoneal or the pleural, are usually the last to be affected. In renal dropsy the face is generally pallid; in cardiac it is dusky, and the surface of the skin is often marked by ecchymosed patches. In this latter condition slight wounds may become serious sores, and the sores sometimes gangrenous. The

most common cause of dropsy in heart disease is imperfection of the valves. Of such, the most certain and most direct is disease of the valve of the right side of the heart—the tricuspid. This disease, however, is comparatively rare; the most common valvular imperfection which gives rise to dropsy is disease of the mitral valve. Aortic lesions, however, sometimes induce the complication.

We now turn from the more general to the more particular points of observation. Of these, first notice the condition of the veins, especially of those at the root of the neck—the internal jugular and the external jugular. Notice if the veins are distended. We have already noticed venous turgescence in the consideration of Cyanosis, and have seen that it is a sign of distension of the right side of the heart. You observe this fulness of veins in conditions of asphyxia—you can produce it temporarily by holding your breath—you observe it in those who play forcibly upon wind instruments, or in patients during the efforts of paroxysmal cough. In these cases there are stasis of blood in the lung, accumulations in the venous channels, and retention in the right cardiac chambers. In a chronic form you may see the same thing in patients affected with emphysema of the lung; in these the right chambers, being in a lasting state of distension, become dilated. Turgescence of the superficial veins may be due, however, to valvular lesion of the right side—to disease of the tricuspid or the pulmonary valves. Sometimes, but very rarely, the veins of the neck may be seen to be varicose. Closely observe the veins to ascertain whether they pulsate. The phenomena of *venous pulsation*, though rare, are of great interest and importance. A very

slight pulsation at the root of the neck may be no more than natural; a slight wave of pulsation in a distended jugular synchronous with the systole of the heart is a sign that the right auricle is distended, and that the contracting ventricle communicates to it its impulse. Be careful to note that the pulsation which appears to be in the vein is really so. The impulse may be conveyed by the artery beneath. To determine this, place your finger on the vein and press the blood upwards for a short distance so as to empty a portion of the vessel; retain your finger thus for a short period; then, if there is distinct venous pulsation, you will see the vein fill from below by jets synchronous with the beats of the heart. In the case of arterial pulsation beneath the vein, the latter does not fill by jets, and you readily feel the strong pulsation of the artery. If you satisfy yourself that there exists venous pulsation, the observation is of high diagnostic import—it means that the tricuspid valve is faulty, and permits regurgitation into the right auricle.

But, perhaps, you perceive not venous, but *visible arterial pulsation.* You notice, we will suppose, that the carotids pulsate forcibly. There may be a vibration over the sternal notch due to the pulse in the aorta. If you look at the situation of the brachial artery in the upper arm, along the inner border of the biceps muscle, you will notice a jerking movement of the vessel at each impulse of the heart. On causing your patient to flex the arm, the movement of the artery is rendered still more pronounced. The vessel is seen to curve outwards from the centre line of the arm with the contraction of the heart, and then quickly return to its first position. Such is the *locomotive pulse* described, in association with the lesion which

causes it, by Sir D. Corrigan, and often called *Corrigan's pulse.* This phenomenon is of great importance, for it is significant of one of the most grave of valvular lesions; incompetency of the semilunar valves of the aorta permitting reflux of blood into the left ventricle. The ventricle, which in these cases is hypertrophied, contracts with an exalted force and drives the jet of blood into the arteries with a sharp and sudden stroke, but, immediately on the subsequent dilatation of the ventricle, the arterial current flows back, leaking through the imperfect valve, and the arteries become abnormally empty. The natural result of the forcible filling of the partially emptied tube is the jerking pulse. You may observe visible pulsation of arteries in cases of aneurism and of hypertrophy of the left ventricle,* but this peculiar jerking pulse is characteristic of aortic regurgitation.

We will now narrow our area of observation to the neighbourhood of the heart itself. First observe whether the beating of the heart causes vibration at any part of the chest-wall or not. The heart-impulse may not be visible for many causes—a thick layer of subcutaneous fat in your patient, an encroachment over the normal situation of the heart by a portion of emphysematous lung, any cause which displaces the heart from the thoracic parietes, or an enfeebled condition of the heart itself. The absence of apex-beat is only of value when taken with other signs, but it is to be noted. Suppose that the beating is visible in one of the intercostal spaces, it is advisable not only to take a mental note of its position, but to mark it upon the cuticular surface with a soft lead pencil, a

* *Vide* subsequent Lecture on "Palpation."

spot of ink, or tincture of iodine, or, if you wish a more permanent record, the stain of a moistened point of nitrate of silver.

The apex, under normal conditions, beats between the fifth and sixth ribs. To determine the spot at which it ought to be evident, you can make one of two observations. (1) Draw a vertical line rather more than an inch internally to the nipple. Where this line intersects, the fifth intercostal space is the spot where the apex, in the average of healthy hearts, is evident. The nipple can be taken as a fixed point in cases of men and children; but not so in case of women. Then (2) draw your vertical line two inches from the left edge of the sternum; where this intersects, the fifth intercostal space is the spot required. Slight variations from this point may be, however, not abnormal. In adult life the apex may approach within a quarter of an inch of the nipple-line, or may recede to two inches nearer the sternum. I think you may take it that in adults, an apex-beat, which is either in the nipple-line or outside it, is abnormal; but in children you cannot conclude that there is abnormality unless the apex is outside the nipple-line. Any deviation, however, from the transverse line of the fifth intercostal space is irregular or abnormal.

Many causes may produce displacement of the apex from its normal position, but such displacement is not usually discoverable by mere inspection. We shall, therefore, briefly consider most of these causes hereafter. The displacement chiefly obvious to inspection is *where the apex beats below and externally to the normal position.* This indicates hypertrophy or dilatation, or both combined, of the left ventricle. In hypertrophy the beating may be observed as far as the eighth in-

tercostal space, or even lower, and it is distinct, strong, and defined. In dilatation it does not usually reach quite so low, and is more diffused and more obvious in the *lateral* direction. In hypertrophy of the right side of the heart there is sometimes marked visible pulsation in the space between the situation of the normal apex and the ensiform cartilage. Note particularly the area over which the visible pulsation is manifest. Usually it is simply a tap in the fifth interspace. In hypertrophy this area is enormously increased, especially in children before the ribs have fully ossified, and the cartilages have become firm. I have observed the vibration in an extreme case of cardiac hypertrophy to extend outwards as far as a line depending from the anterior border of the axilla downwards, to half way between ensiform cartilage and umbilicus, and to the right to a considerable distance beyond the right border of the sternum. In cases of considerable dilatation and hypertrophy the movement of the chest-wall caused by the cardiac contractions may be seen to be undulatory. It is obvious that the motions of the muscles which contribute to form the heart are not synchronous. Pulsations may be seen over the situation of a dilated auricle right or left. We shall learn in the next lecture that enlarged and hypertrophied auricles can give rise to pulsations which differ in point of time from the ventricular contractions. This is one cause of the apparent undulation. Adhesions of the pericardium may increase the irregularity of the movement.

The area of visible impulse may, however, be considerably increased, although there be no hypertrophy; in nervous palpitation, in some cases of anæmia, in Graves' disease or in chorea, pulsation

may be observed over a space the size of the palm of the hand. You will note, however, that in such cases, the apex-beat is *not displaced* from its normal position, nor is the chest-wall bulged outwards.

You will notice particularly whether the præcordial region is rendered unduly prominent. The bony chest may be made thus prominent by rickets. You will recognise this condition by the fact that other portions of the thorax present nodosities, bulgings, or irregularities. Carefully observe whether there is any spinal curvature, for this, by causing depression of the thorax posteriorly, may induce bulging in front. If you are satisfied that there is distinct prominence of the heart region you will notice whether there is pulsation over the area—if so, you have obtained another evidence of hypertrophy. If, however, pulsation is feeble or absent, and the intercostal spaces are rendered convex, the presumption is strong that there is effusion into the pericardium. If the intercostal spaces are retracted so as to be rendered concave when the heart contracts there is a probability that pericarditis has at one time existed, and has resulted in adhesions of the pericardium. In some cases this retraction with the cardiac systole is manifest in the epigastrium to the left of the ensiform cartilage. This is due to the attachment of the base of the pericardium to the central tendon of the diaphragm.

We may now recapitulate briefly the evidence which we have obtained by inspection in relation to its diagnostic value. We have obtained evidence which indicates the highest point of probability in cases of (1) cardiac malformation, producing cyanosis, (2) Graves' disease. (3) distension of the right side,

(4) incompetency of the auriculo-ventricular valve of the right side (tricuspid), (5) incompetency of the semilunar valves of the aorta, (6) hypertrophy of the heart, (7) dilatation of the chambers of the heart. We have gained a considerable amount of information leading to the diagnosis of (8) acute pericarditis in cases when effusion has taken place in sufficient amount to obviously distend the pericardium, and of (9) a past pericarditis when pericardial adhesions have taken place in any considerable degree. We have obtained valuable evidence though in less degree, of (10) conjunction of renal with cardiac disease, (11) fatty degeneration of the heart, and (12) valvular lesions where they have given rise to symptoms of dyspnœa.

We next propose to exercise the sense of touch to form or confirm our diagnosis.

LECTURE III.

PALPATION.

The pulse—radial compared with cardiac—Rhythm—Effect of effort—Position of apex-beat—Displacement—Topography of heart—Mechanism of auricles and ventricles—Chronometry of cardiac pulsations—Presystolic, epigastric, and hepatic pulsation—Pericardial friction-fremitus—Thrill.

We have now to cause our perceptions to enter by the tips of the fingers; we gain our knowledge through the sense of touch.

We will first examine the *pulse*. It is better to do so now that the chest, neck, and upper extremities are exposed, in order that we may consider the radial beats in relation with any obvious pulsations in arteries elsewhere.

(*a*.) Notice whether there be any *peculiarity* in the radial artery. It may be rigid, unyielding, presenting irregular hard plates, as it were, embedded in its wall. This indicates *atheroma*, a degeneration or calcification of the coats of the vessel. Your patient, in this case, will be past the prime of life, for the condition is essentially senile. The pulse will be felt to be hard and strong, the left ventricle having become hypertrophied, and contracting forcibly to overcome the obstruction created by the inelastic arteries. The changes of the radial are indicative of like changes in many other arteries of the body. You may probably observe the temporal artery

tortuous to the eye, and rigid to the feel, its pulsations visible. Frequently there is a semblance of strength in the pulse without reality, the shock being created by a feeble wave of blood in a rigid tube; in such case you will find that the impulse at the apex of the heart is feeble; this indicates that the atheromatous condition of the arteries is associated with fatty degeneration of the heart.

(*b.*) Observe in the next place whether there is a difference in volume and force between the pulses of the two radials. If so, examine the brachials; if these are equal, the difference is merely one of irregular distribution. If there is a decided difference in the pulses of the upper extremities, there is a probability of aneurism of one of the great vessels.

(*c.*) Eliminating the above conditions, we now examine the pulse in relation to rapidity, regularity, strength, and volume.

(1.) Notable *slowness* of the pulse is generally due rather to neurosal than to cardiac causes; it may occur, however, in a heart extremely weak from fatty degeneration. The pulse has been noted as low as 20–30, and even 8–9 per minute.

(2.) *Rapidity* of pulse is much more common. It may be found associated with pyrexial conditions in the early stage of pericarditis, before effusion to any amount has taken place in the sac. Very frequently, however, abnormal quickness of pulse is not noticed at any stage of pericarditis. Of very far greater significance, is a point noticed by Dr. Walshe—*sudden variation of the rate* of the pulse. In a case of pericarditis, a very slight movement of the body may increase the pulse from 80–90 to 120–140. Unusual quickness of the pulse occurs in many emotional

conditions, and you must not rely upon it, except in connexion with other signs.

(3.) *Force of the pulse.*—This is a point of great moment. The apparent force of the pulse is notably *increased* in two conditions—hypertrophy of the heart and regurgitation through the aortic valves. It is *diminished* in degeneration of the muscular fibre of the heart, in dilatation of the cavities, and in most of the valvular affections.

In hypertrophy we find the pulse strong, full, and incompressible. As you feel the radial pulse with one hand, place the other upon the thorax over the heart-region, and you will find a strong heaving, prolonged cardiac impulse as an accompaniment. There is an expression of power, both about pulse and apex-beat, which is quite wanting in simply functional excitement of the heart. In the latter case the stroke is not sustained, but abrupt and brief.

The other cause of exalted force of pulse, aortic regurgitation, has already been indicated to us by signs which we have considered under "Inspection." The pulse is first jerking, then collapsing; the artery strikes the finger with a sudden blow.

Suppose now that, on the other hand, you find the pulse small and feeble, *it is a good rule to elevate the patient's arm vertically above his head, and observe the characters of the pulse in this position.* This will enable you to eliminate doubtful cases of aortic regurgitation in which the pulse has become temporarily feeble; in the aortic regurgitant lesion, the pulse will become intensified instead of enfeebled, its hammer-like character exalted, and probably the patient will complain of discomfort. In most of the other

valvular lesions, aortic obstructive disease excepted, in which no influence probably will be detected, the pulse is enfeebled by the vertical position of the arm. In some cases of mitral disease, and in conditions wherein the heart-muscle is feeble, the radial pulse in this position may be quite extinguished. The same occurs, however, equally in anæmia. The pulse being perceptible, notice whether the vertical position induces irregularity, especially irregularity of *volume*. If so, there is a probability of disease of the mitral valve.

You should now, while still keeping the finger upon the radial pulse, examine with your other hand the situation of the heart's impulse. It is very important if you note that, though the heart's contraction is strong, the pulse at the wrist is small and weak. This suggests imperfection of the mitral valve, inducing either regurgitation or obstruction. In mitral regurgitation the current of blood which should, by the contraction of the ventricle, be forced into the aorta, and thence to the systemic arteries, is, by the leak in the mitral valve, in part diverted to the left auricle. The pulse, therefore, is not proportionate to the strength of the heart's contraction, but is in inverse ratio to the amount of blood lost to the arteries by regurgitation. In mitral obstruction, the pulse is weak because the blood reaches the left ventricle with difficulty, and the latter contracts on an insufficient amount. It is not often possible by mere observation of the pulse to differentiate between obstructive and regurgitant lesions at the mitral orifice, but notable irregularity in volume and rhythm points with greater probability to regurgitation. In the former case, dilatation of the ventricle does not

occur, and the muscle is not enfeebled; in regurgitation, dilatation of the cavity is most common. In many cases of mitral regurgitation, the pulse appears to be quite normal. This indicates either that but a small quantity of blood is lost to the systemic arteries by such regurgitation, or else that the left ventricle has become hypertrophied sufficiently, and contracts with adequate force, to compensate for the obstruction caused by the reflux into the auricle. If you notice the pulse to be persistently weak, the contractions of the heart feeble, and yet by examination you find no evidence of valvular disease, there is a strong probability that the heart-muscle is enfeebled by degeneration.

If you find that the pulse is feeble, although the hand, placed over the præcordium, detects tolerably strong pulsation, and yet evidence of valvular disease is absent, then it is very probable that the hypertrophy of the heart causing the strength of systole is in the *right* ventricle, and not in the left.

Occasionally you may find that the hand on the præcordial region is sensible of a contraction, which does not make itself evident by a pulse at the wrist—there is an *ineffectual systole.* The ventricular contraction is at such time too feeble to produce a sensible pulse in the remote arteries. It will be found, however, that, though lost in the radial, the pulsation can be detected in the larger arteries nearer the heart, such as the carotids. This is but a less significant expression of the conditions we are about to consider—viz., *irregularity* and *intermittency.*

(4.) *Rhythm of the pulse.*—Carefully distinguish between *irregularity of volume* and *irregularity in time.* In the former case the pulse is felt to occur at equal

intervals, but is fuller at one beat than at another. You are sensible that varying volumes of blood are transmitted by the various contractions of the ventricle. This condition is rendered more evident by elevation of the arm, and the observation may give you an important aid to diagnosis. It is almost pathognomonic of mitral regurgitation.

We will suppose that the pulsations are irregular in time. Irregularity and intermittency are conditions which differ only in degree, so we will here consider them together. In intermittency, the pause between the pulses is longer, there being an interval equal to that occupied by a pulsation. The first thing I have to urge upon you is to be very careful in giving this phenomenon its due weight in a diagnostic sense, and no more. I am afraid that mischief has been done many times by a hasty opinion to the effect that a patient has heart-mischief, when this sign has been too exclusively relied on. Remember that irregularity or intermittency of heart's action, and consequently of pulse, may coincide with organic integrity of heart, and even with good health. The first question I would ask you to propound to yourselves is :—Does this irregularity co-exist with a fair strength of impulse, as felt over the præcordium or otherwise?

Intermittency may be merely a constitutional peculiarity. It may be due, as Dr. B. W. Richardson has shown, to causes operating upon the general nervous system. It may be superinduced by strong emotions, by terror, anxiety, grief, pain, fatigue; it may occur in organic diseases of the brain. It may be temporarily caused by attacks of indigestion. In the absence, therefore, of a notable feebleness of heart-

beat, and excluding other diagnostic signs, you are not to conclude from mere intermission of heart and pulse that organic cardiac disease exists.

If, however, notable feebleness of impulse co-exists with irregularity it is of serious diagnostic import. It is the sign, the very early sign it may be, of a strike on the part of the left ventricle. Too languid to contract from the ordinary stimulus of the blood with which the auricle by its single contraction supplies it, it waits until a second, or even a third, contraction of the auricle has supplied it with more. This is the condition which obtains in mitral regurgitation and in dilatation of the ventricle. It is pathognomonic of dilatation of the left ventricle, and indicates that the muscular fibres have become degenerated, and have lost their tonicity. Most commonly it is associated with mitral regurgitation, which is the frequent inducing cause of such dilatation. When you observe in a case of mitral disease that the heart's action assumes this character, note it as a sign of prognostic import. In the absence of signs of valvular disease the observation is of importance, both from a diagnostic and a prognostic point of view. When you find it in conjunction with senile changes—with feeble but diffused impulse, with atheromatous vessels, "arcus senilis" of cornea, &c., you may know that a heart at one time hypertrophied has become degenerated, that it will never recover its power, and that the patient trembles on the edge of life.

A further question I would ask you to propound to yourselves when you notice in any patient irregularity or intermittency of heart's action is:—What is the effect of effort upon this heart?

An irregularity which is merely neurosal is scarcely affected by effort; the pulse is quickened of course, but its irregularity is often diminished instead of increased. When the irregularity is due to cardiac imperfection, however, very slight effort, such as making your patient walk briskly for a minute or two up and down the room, notably increases such irregularity. When there is dilatation, palpitation usually precedes the more pronounced irregularity; but when degeneration has proceeded far, the halting action takes place without notable quickening.

Notice also whether effort on the part of your patient causes distress, and what is the form of such distress. Little or no discomfort occurs in neurosal irregularity. In dilatation you find palpitation and dyspnœa; in degeneration, faintness and dyspnœa; in certain rare cases you find that the ventricular halt is accompanied by horrible sensations, and a fear of impending death. Such was noticed by Romberg in a case in which a tumour involved the vagus nerve.

We turn from the consideration of the pulse to that of the cardiac area itself.

We have noted in our inspection of the chest where the impulse of the apex of the heart should be manifest. We proceed to confirm or enlarge our observation by placing the hand over this region. Suppose that

(*a.*) *The apex-beat is feeble or indistinguishable.* Before recording this as a positive observation, let your patient sit up and lean well forward; an apex-beat may then become evident which was before undetected. If notable feebleness of the beat be associated with weakness of the pulse and the signs we

have before recorded as pertaining to the affection, you have gone very far towards the diagnosis of fatty degeneration of the heart. In rare instances, such feebleness may be found to be due to pericardial effusion; in such case the postural change just noted will serve you in good stead as an additional means of diagnosis, for the tilting forwards of the body may render evident the apex pulsation in a situation above the normal, probably in the fourth intercostal space, the fluid in the pericardium having tilted the apex upwards to this level. Palpation is of high importance in the diagnosis of pericarditis with effusion. In the earlier stages of the affection you may find excited, diffused, and, sometimes, tumultuous, action of the heart with the general signs of pyrexia; as effusion takes place the apex-beat is enfeebled and carried upwards, and to the left of its normal position; then, if distension be extreme, the beat is no longer to be felt. You must, however, draw no hasty conclusion from the observation that the apex-beat is feeble or imperceptible to the touch. It may be thus because the heart is overlapped by emphysematous lung, or on account of a thick layer of subcutaneous fat in your patient, or, even in health, in persons with deep chests. You may readily understand that in women with full breasts the apex-beat may not be perceptible to the touch. It is a fundamental rule, however, that you should always observe in cardiac diagnosis—*to fix the exact spot of the apex-beat.* If inspection and palpation fail to do this, you will proceed to determine it by auscultation, as we shall hereafter consider.

We will now suppose that you have been able to feel the apex-beat, and have compared its position

with that which it should normally occupy, as we have determined in the lecture on inspection. You find that

(*b.*) *The apex-beat is displaced from its normal position.* Such displacement may take place from disease of the neighbouring textures and organs. Pleuritic effusion in the left cavity of the thorax may push the heart completely to the right side, so that the impulse which is wanting on the left side is felt right of the sternum; effusion in the right thoracic cavity, on the other hand, may push the apex left of its normal position. Emphysema of the lung pushes the heart downwards and towards the epigastrium, and oftentimes in this disease you find an impulse below the ensiform cartilage; you must examine further, however, before concluding that this is the apex-beat, for in emphysema the right side of the heart is usually enlarged, and the impulse which you feel is caused by the contraction of the right ventricle, the tip of the left ventricle being outside the point of obvious pulsation. Another disease of the lung which may give rise to a singular displacement of the apex-beat is fibroid phthisis; in such condition, when it affects the left lung, you may find the chest-wall drawn inwards and the heart so displaced that its apex beats at or above the fourth rib. Tumours, aneurism, or cancer occupying the thoracic cavity may also cause displacement of the heart: enlargement of the left lobe of the liver, cysts, abdominal tumours, and dropsy may produce a like result. We have already said that pericardial effusion tilts the apex upwards. All these causes must be eliminated by careful examination. We come now to causes of displacement intrinsic to the heart itself.

When the heart-apex is found to beat below and to

the left of its normal position (fifth interspace and two inches from left border of sternum, or an inch and a half right of a vertical line through the nipple), you may diagnose hypertrophy of the muscular wall of the left ventricle or dilatation of the ventricular cavity. The sensations communicated to your fingers aid you to differentiate those two conditions. Remember that *hypertrophy* means *power*, and *dilatation weakness*, but the phenomena and their causes may be variously combined. A full, long, and heaving stroke is characteristic of hypertrophy; an excited, short, diffused, struggling beat indicates dilatation. Action with power is shown in the one case, excitement without power in the other. In hypertrophy and dilatation the apex may be felt two or three inches, or even more, outside the nipple-line, and as low as the seventh or eighth intercostal space. If the apex happen to beat against a rib the shock is, of course, subdued; it is necessary to recollect this, otherwise you might put down as feeble an impulse which is really strong.

We now consider not only the *situation* of the tactile pulsation, but the *extent* of area over which it is manifest. Normally, as I have said, only a tap in the fifth interspace is felt; pulsation may be evident, however, over a superficial inch in strictly normal conditions. Pulsations in other positions may possibly be evident in health. Such may be felt in slight degree in the fourth intercostal space; or, and this more commonly, in the epigastrium to the left of the ensiform cartilage. By pushing the fingers upwards beneath the false ribs on the left side, you may sometimes feel the throb of the right ventricle. You must give these contingencies due weight, but, as a general rule, any pulsation

manifest in other situations than the neighbourhood of the normal apex, is evidence of disease of the heart or the great vessels. We proceed to consider the conditions wherein

(*c.*) *Pulsations, apart from the apex-beat, are manifested over the cardiac area.* To appreciate these we should endeavour to obtain an idea of the normal TOPOGRAPHY OF THE HEART. The area occupied by the healthy heart may be thus roughly illustrated on the thoracic wall :—Draw a line, a little externally to the right border of the sternum from the second intercostal space to a point just below the fifth sterno-costal articulation. Draw a second line from a point just below the second sterno-costal articulation on the left side to the situation of the normal apex. Unite the extremities of these lines respectively above and below, so as to describe a quadrilateral figure. Of this superficies (which will be bounded above by the aorta which crosses from right to left, and below by the diaphragm) about four-fifths are occupied by the right ventricle, which lies immediately behind the sternum and the third, fourth, and fifth costal cartilages of the left side, and culminates in the pulmonary artery at the second (left) interspace close to the sternum. The left ventricle is chiefly posterior; but it borders the right ventricle to the left from the third sterno-costal articulation to the apex, which itself constitutes. The right auricle is in the third interspace (right) and behind the third and fourth cartilages. The left auricle is for the most part overlapped by the pulmonary artery, but a small part of it is situated superficially in the second interspace left of the sternum.

If you find a forcible impulse of the heart towards

the left of the border we have sketched as the normal left boundary of the cardiac area, the apex strongly defined in its beat and manifest below and to the left of its usual place, the pulse being full and strong, you may conclude that there is hypertrophy of the *left* ventricle. If you find a strong impulse in the epigastrium extending from the normal apex to the right of the ensiform cartilage and sternum, and yet a much weaker pulse than you would think such a systole would produce, you probably have hypertrophy of the *right* ventricle. Tuck your fingers under the false ribs to the left of the ensiform cartilage, and you will feel the contraction of the ventricular wall. We shall return hereafter to epigastric pulsations.

As I have before said, hypertrophy and dilatation are often combined. When the left ventricle is dilated as well as hypertrophied, palpation gives you a less localised and less firm and strong impression. The impulse is more diffuse, the apex feels less pointed, and more rounded or globular to the finger.

When the left ventricle is dilated, but not hypertrophied, you feel its impulse over a wide area in the situation indicated, but it is felt as a short excited "slap."

Hypertrophy of the right ventricle scarcely ever exists without dilatation.

It has been supposed that auricular hypertrophy is not to be demonstrated by physical signs. I shall presently show you that this is not the case, for I have in many instances been able to demonstrate upon the surface of the chest-wall the contraction both of the right and of the left auricle.

When you have a general dilatation, or hypertrophy and dilatation combined, of all the cavities of the

heart, the hand and the eye both perceive a fluctuation—or, as Dr. Walshe has expressed it, a seeming undulation—over the extended area over which the contractions of the cardiac chambers are manifest. This is obviously due to the fact that the contractions of the muscular walls of the various chambers are occurring not simultaneously, but in successive moments of time.

We will briefly consider the mechanism of the muscular walls of the heart. We should remember that the auricles and ventricles possess the double function of *reservoirs* and *propellers*. Such function is exercised in alternation—at rest they are reservoirs, in action they are propellers. You know that the rhythm of the heart comprises a period of contraction (systole), and a period of rest and dilatation (diastole). The latter occupies much the longer time: as a rule, the systole occupies one-fifth of the period; the diastole, of course, the remaining four-fifths. During the period of repose, what is taking place? On the right side of the heart, the great veins of the body, the *superior vena cava*, and the *inferior vena cava*, are pouring their impure venous blood into the right auricle, which is gradually filling; the ventricle is becoming filled at the same time, for, the auriculo-ventricular valves being now flaccid, auricle and ventricle form one cavity. On the left side of the heart, the pulmonary veins are carrying the pure blood, which has been aërated in the lungs, to the left auricle, and hence to the ventricle, these cavities becoming filled simultaneously with the right. During all this period, the muscle of auricles and ventricles is receiving its nutrient supply, for the heart differs from all the other structures of the body in that, whilst the latter are

supplied with blood by the heart's contraction, itself receives its arterial supply during its own period of repose. The muscle of the heart is supplied with blood through the coronary arteries, which arise from the aorta (pouches of Valsalva) just above the semilunar valves. The open mouths of these arteries had just been occluded by the flaps of the valves whilst the aortic orifice was open, but now in diastole, the valves falling back from the weight of the superincumbent column of blood and closing the aortic aperture, the channels are opened for the sudden in-rush of the stream of arterial blood into the tissue of the heart.

The cavities having become replete, the contraction of the heart, the systole, begins. The important point to notice is, that this contraction is not synchronous throughout all the muscular cavities of the heart; but that *the auricles always contract before the ventricles.* You are aware that the fibres which constitute the muscle of the heart are under the control of the minute ganglia of the sympathetic nerve; the action of these ganglia is, however, controlled and co-ordinated by the vagus nerve, which restrains contraction until such contraction shall be uniform and regular. The necessary stimulus to the contraction of the auricle is *distension;* until it is sufficiently filled with blood, its systole does not occur. Distension, then, provokes auricular contraction, and the ventricle which has already been filling by the passive flow of blood into it, is now more completely gorged by the additional blood forced into it by the auricle. In the normal heart, this precedence in action of the auricle is only momentary, the wave of contraction speedily ensuing in the ventricle. Still, it is of high importance to

realize this priority of auricular contraction. Though the arrangement of the muscular fibres of the heart is such, that many of the fibres are common to all the cavities, yet the auricles are much more independent of the ventricles than the ventricles are of each other. Extremely early in fœtal life there is a mark of differentiation of the then single auricle from the single ventricle, but the right ventricle is formed by a doubling over of a part of the original (left) ventricle.

The necessary stimulus, then, for the contraction of the ventricles, is the extra repletion induced by the immediately preceding contraction of the auricles. The ventricular contraction, though strictly speaking vermicular, occurs in such a brief moment of time, that it is indistinguishable save as one movement in synchronism. The simultaneous contraction of the ventricles driving the blood into the aorta on the one side, and the pulmonary artery on the other, the apex-beat, the pulse in the aorta, and the pulse in the pulmonary artery, are practically coincident in time.

I have said that in the normal state of the heart, the pulsation of the auricles causes no apparent vibration of the thoracic wall; it is not so in disease, however; hypertrophy and dilatation of right or of left auricle may give rise to a visible pulse. As regards the *right* auricle, this has been generally admitted. The right side of the heart becomes dilated, and often hypertrophied, under all circumstances which induce an undue repletion of the venous system. We have already alluded to these conditions, and we shall again briefly consider them in the next lecture. The right auricle is never hypertrophied and dilated without the right ventricle being in like con-

dition. Under such circumstances you feel a heaving impulse, which seems to be very near the surface, extending from the normal apex across the epigastrium to the right side of the sternum; this is caused by the right ventricle, and you may feel a pulsation in the second or third intercostal space just right of the sternum, which is caused by the auricle. How to determine this we shall presently consider.

We turn now to the *left auricle.* Undoubtedly, in certain cases, you may observe a pulsation in the second or third intercostal space left of the sternum, but is this caused by the auricle? On this point there has been difference of opinion. Some writers have stated that inasmuch as the greater part of the auricle is covered by the great arteries emerging from the heart, and the muscular walls of the auricle are comparatively thin and feeble, its power of contracting with sufficient force to produce an impulse is improbable. One of the most recent writers on heart disease, Dr. Milner Fothergill, has said that "the left auricle can scarcely be said to have any physical signs connected with it." These observations are undoubtedly correct in reference to the left auricle when in a state of health. It is a very different matter, however, when it is in a state of disease. There is one condition of disease or malformation, which gives rise to great hypertrophy and dilatation of the left auricle—*narrowing* (*stenosis*) *of the auriculo-ventricular orifice of the left side* (*mitral*). The causation of this hypertrophy and dilatation is easy to understand. I have explained that the contraction of the auricles is necessary to produce that perfect repletion (that additional supply to the already partially filled ventricle) which is required to call forth the

ventricular systole. When the aperture between auricle and ventricle is so narrowed as to become an obstruction, the auricle has perforce to contract with enhanced power to overcome it. The auricle thus called upon for abnormal force of contraction becomes hypertrophied, and, from being in a state of preternatural distension, dilated. In the case of a boy of nine, who had constriction of the mitral orifice, I found at the post-mortem examination that the muscular wall of the left auricle was from $\frac{1}{8}$ to $\frac{1}{4}$ inch in thickness.* The normal thickness of the left auricle in adult life is somewhat more than $\frac{1}{12}$ inch.† According to Bouillaud, it is $\frac{3}{20}$ inch. Here was an auricle, the thickness of the muscular wall of which was in great part thicker than the thickest part of the normal adult right ventricle. Who could doubt the ability of such an auricle to communicate a distinct pulsation to the thoracic wall?

The foregoing considerations will point us to a plan of demonstrating such pulsations as are caused by the auricles. A pulsation left of the sternum may be due to the contraction of the left ventricle, the pulse in the pulmonary artery, or the contraction of the auricle. A pulsation right of the sternum may be due to the contraction of the right ventricle, or the right auricle, or the pulse in the aorta, or some aneurismal dilatation of that vessel. To determine whether the pulsations are, or are not auricular, we may adopt a simple plan of CHRONOMETRY OF PULSATIONS OCCURRING OVER THE CARDIAC AREA. Dr. George Balfour, of Edinburgh, has recommended

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* *Medical Times and Gazette*, January 10th, 1874, p. 35.

† Flint, "Diseases of the Heart," 2nd edition, p. 21.

that the movements of two doubtful points of pulsation should be compared by attaching to each, by means of a pellet of beeswax, a bristle carrying a small paper flag. The variation in time of the two movements is observed by the vibrations of the flag. The modification of this valuable plan which I adopt, is the following:—Cut two small circles of sticking plaster, about the size of fourpenny-pieces; transfix the centre of each by a pin, so that the head is in contact with the adhesive side of the plaster. Attach the one adhesive circle over the site of pulsation supposed to be auricular, and the other over the situation of the apex of the heart. The shafts and points of the pins projecting forwards, you have two levers which vibrate with the movements communicated to them by the several pulsations; these levers you elongate by attaching to them rolled "spills" of tissue paper.

If you have to do with an auricular pulsation you will see that the movement of its lever invariably *precedes* that of the lever adapted over the heart's apex. The movement is *presystolic*, and must be due to the contraction of the auricle. If the two contractions occur simultaneously they must be produced by the ventricle or in the pulmonary artery or aorta.

Pulsation of the pulmonary artery, which may be felt between the second and third ribs, close to the left border of the sternum, is only manifest when from any cause the left lung is retracted from the base of the heart. You feel that such pulsation is very superficial; it is said that even the click of the pulmonary semilunar valves may be felt as a little shock to the finger. It is interesting to note the

relation of this phenomenon to respiration. If the lung be only partially retracted from the pulmonary artery, you will observe the pulsation to become more and more visible during *expiration*, whereas, during inspiration, as the lungs becoming more and more filled with air encroach over the heart, the pulsation becomes less and less evident till it ceases, reappearing at the next inspiration.

Pulsations of the aorta, which, of course, occur on the opposite—the right—side of the sternum, may be due to aneurism, or to displacement of the vessel which occasionally occurs in rickety chests.

It remains for us to consider *pulsation felt at the epigastrium.* We have already noticed the pulsation due to a displaced or hypertrophied and dilated right ventricle. Any considerable enlargement of the right side of the heart will give rise to a pulsation felt at the epigastrium. An impulse is sometimes communicated to the edge of the left lobe of the liver, which is to be felt below the ensiform cartilage. Occasionally, but very rarely, the impulse is *reversed;* the integuments at the epigastrium are felt to be *retracted*, instead of propelled at each systole of the heart. In such cases there has been an extensive pericarditis, which has resulted in adhesion of the heart to the diaphragm and the liver.

But we have to consider pulsations felt at the epigastrium, which may be due to other causes, with a view to differential diagnosis. We will suppose that:

(*A.*) The pulsation is felt in the median line, that is, the line joining the ensiform cartilage and the umbilicus. It will probably occur to you that such pulsation may be due to aneurism of the abdominal

aorta. The suggestion readily occurs, but though you feel a forcible pulsation over the vessel, you must hesitate very considerably before committing yourself to the opinion that it is due to an aneurism. Abdominal aneurism is rare. "So," as Sir William Jenner has said, "instead of being your first, it should be your last idea, that an abdominal pulsation is due to aneurism."* If there be an aneurism, you will feel a localised swelling of the vessel, which with, or just after, the systole of the heart expands equally in all directions, above, below, and laterally. A tumour, superficial to the aorta, may pulsate from the communicated impulse of the vessel; but then you will feel no lateral pulsation. You may find assistance in the diagnosis, by causing your patient to bend forwards on the hands and knees; a tumour isolated from the vessel then recedes from it, and you no longer feel the pulsation, which in aneurism is unaffected by this position. Having eliminated abdominal aneurism and tumours to which the abdominal aorta may communicate its impulse, we will suppose that you yet find a pulsation in the central line, below the ensiform cartilage, which you have convinced yourself, by palpation under the false ribs, is not due to the impulse of the right ventricle. You will find such a pulsation very commonly; it is the pulse of the abdominal aorta. In the vast majority of abdominal pulsations there is no structural alteration to account for them; they are palpitations due to neurotic conditions. The abdominal aorta shows the excited pulse that the other great arteries

* Clinical Lecture on Tumours of Abdomen: *British Medical Journal*, 1869, p. 42.

of the body manifest. This phenomenon is, of course, most evident in spare people; you will find it in cases of anæmia, in dyspepsia, and especially the dyspepsia of old people whose arteries have commenced to degenerate. We will consider in the next place that:

(*B.*) The pulsation is felt to the *right* of the median line. I have said that the beating ventricle may propel the edge of the left lobe of the liver; an aneurism may do the same still more extensively; but in some cases it may be observed that *the whole liver pulsates.* This phenomenon is of rare occurrence, but it is of great diagnostic importance: it indicates regurgitation through the tricuspid orifice. The liver, in this condition, is like an erectile tumour; it pulsates with the systole of the heart. The right ventricle, by its contraction, instead of driving all the blood it contains into the pulmonary artery, on account of the imperfection of the tricuspid valve, forces some of its contents back again into the right auricle. Consequently, an impulse is given to the blood contained in the vessels opening into the auricle—*i.e.*, the great systemic veins and their tributaries. Hence the rhythmical injection of the hepatic veins, and the consequent pulsation of the liver. You will see that this phenomenon is exactly analogous to the venous pulse in the jugulars which we have before noticed. Both indicate the same morbid condition of the heart, tricuspid insufficiency; but their relative value, in a diagnostic point of view, I cannot consider to be decided. Some Continental observers will have it that the beat of the liver is one of the *earliest* signs of the lesion, a sign preceding, in the course of disease, the pulsation of the jugulars. I do not think that

our experience bears out this view, but there can be no doubt that the phenomenon, when observed, is a crucial sign of insufficiency of the tricuspid valve. Suppose, now, that:

(*C.*) The pulsation is felt to the *left* of the median line. This may be due to an aneurism of the abdominal aorta, especially when it involves also the superior mesenteric artery. I once met with a case in which there was pulsation felt to the left of the middle line, midway between ensiform cartilage and umbilicus. The pulsation gave rise to distress, and was associated with a fulness at the epigastrium. The case had been diagnosed as one of cancer of the liver. Whilst, however, it was evident that the left edge of the liver encroached over the epigastrium, I could find no signs leading to the conclusion that there was any malignant disease. The left side pulsation was very difficult of explanation. I had the opportunity, however, of seeing the case several times, and I found (1) that the pulsation always became much pronounced just previously to the catamenial period (the patient, a woman of full habit, was nearing the climacteric), and that it diminished almost to extinction after the period was passed; (2) that it always was controlled, even to almost extinction, by large doses of quinine. The view that I took of the case was, that the pulsation was in the splenic artery. The patient completely recovered.

We have hitherto considered the evidence afforded by the well-known phenomenon, *pulsation*, as detected over the cardiac area. Now we have to consider other peculiar diagnostic signs recognisable to the touch.

THE SPECIAL TACTILE PHENOMENA MANIFEST

OVER THE CARDIAC REGION are briefly, (*a*) friction, (*b*) thrill.

(*a.*) On placing the hand over the præcordium, you may, in certain cases, detect a sensation of rubbing accompanying the systole and diastole of the heart. You should cause the patient to hold his breath so as to convince yourself that the sensation is not communicated by any part of the respiratory mechanism. If you feel that the periods of contraction and dilatation of the heart are accompanied by this feeling of friction, you may be sure that your patient is suffering from pericarditis. The phenomenon is termed *pericardial friction-fremitus;* it is not common; in a very large proportion of cases of pericarditis it is not observed. There must be the concurrence of certain conditions to produce it. The pericarditis must be in an early stage; it occurs only at the commencement of effusion into the pericardial sac: the fluid effused must be thick or rich in fibrine; at autopsies in some cases of pericarditis, you may see the surface of the heart covered by a layer of material like soft butter, this is the kind of effusion which gives rise to the feeling of friction: the amount of fluid must be limited; as the pericardial sac becomes distended the exudation is less viscid, the heart is separated from the thoracic wall, and the systole is enfeebled—all these causes concur to prevent the sensation of pericardial fremitus: lastly, the heart must contract with sufficient force; a feeble heart does not produce it. This sign never occurs without the friction being recognisable by the ear, as we shall hereafter describe. You will find a great number of cases of pericarditis occurring without this sign, but where you observe it it is of great importance; for it renders certain the diagnosis

of pericarditis, and it enables you to state that the pericardial surfaces are roughened, or that the effused material is of a viscid character.

We come now to an interesting phenomenon, which will well repay careful investigation. This is:

(*b.*) Thrill. On placing the hand over the præcordium you are sensible of a peculiar vibration occuring over a certain area and at a certain period of the heart's action. The vibration is quite characteristic; a sense of trembling is communicated to the fingers—rapidly as if the finger touched the twanged string of a violin, or comparatively slowly as if the vibration had proceeded from the bass string of a violoncello. The French observers aptly compared the sensation to that experienced when one places the hand on the back of a purring cat or kitten; they gave to the phenomenon the name of *frémissement cataire.* It is characterised also by the terms *purring tremor*, and in German, *Katzenschnurren.* It is not sufficient, however, merely to note the existence of this sign. You must especially establish, (1) its position; (2) its rhythm.

You may feel it over the base of the heart, about the second intercostal space and right of the sternal border. Placing the tips of the fingers over the situation where the thrill is felt, now touch with the fingers of your other hand the spot where the heart's apex beats. This will enable you to determine the rhythm of the thrill. You find, we will say, that the thrill coincides in time with the apex-beat. Then you have either contraction (stenosis) of the aortic orifice or aortic aneurism. You will notice whether the latter condition is indicated by a pulsatile swelling or the concurrent signs of aneurismal dilatation of the

aorta. If not, you will proceed to confirm your diagnosis of aortic stenosis by auscultation and the means we shall hereafter describe. In rare cases the purring tremor may be felt over the situation of the pulmonary artery; it indicates obstruction of that vessel. Perhaps, however, you find that the thrill does not coincide with the beat of the apex but occurs during the diastole of the heart. A diastolic thrill is rare; it is characteristic of regurgitation through the aortic orifice. It signifies that the aortic valves cannot perfectly close, that they permit reflux of blood. So you may find this sign in conjunction with the phenomena we have noted as occurring in a like condition — forcible apex-beat and locomotive (Corrigan's) or water-hammer pulse.

Suppose, however, that you feel a thrill at or near the apex of the heart. In this position it is of the utmost importance to establish the rhythm of the thrill. I always advise that for this purpose you employ the fingers of each hand, as in the case of aortic thrill. First determine the spot at which the apex beats. You may find that this is also the point of greatest intensity of the thrill; to assure yourself of this, however, place a finger of your other hand a little externally. You will then convince yourself whether thrill and impulse occur together; in other words whether the thrill is systolic. If so, if thrill = impulse, it is caused by regurgitation through the mitral orifice. Such a thrill is not common. You may find, however, and that much more commonly, that the thrill felt in the neighbourhood of the apex is not exactly simultaneous with the systole. You distinctly recognise that the finger which receives the impression of the thrill does so just before the

finger of the other hand receives the impression of the impulse of the ventricle. The thrill is abruptly terminated by the ventricular systole—it is *presystolic.* Moreover, you may find that it is felt not just at the apex, but slightly internal to it, and at a higher level. This separation of thrill from apex-beat, renders the detection of the rhythm of the former more easy by the rule which I have laid down. *Always investigate the phenomenon of thrill by using both hands, examining the site of the thrill by the finger or fingers of the one hand, and noting the apex-beat by the finger or fingers of the other.* It will be found that of thrills about the apex, that which is presystolic in rhythm is by far the more common. To recognise it is of very great importance. It is pathognomonic of contraction (stenosis) of the mitral orifice, and is due to the vibration caused by the forcible contraction of the auricle urging the blood through the obstructed outlet into the ventricle. We shall return to this subject when we come to auscultation, and consider the relation of thrill to murmur, but it is necessary to premise that both thrill and murmur are due to a like cause, but that we may have thrill without murmur and murmur without thrill. Vibration produces both phenomena; if the vibrations be insufficiently rapid they cannot be perceived by the ear, whilst they are obvious to touch; again, they may be so rapid as to be undetected by touch but detected by the ear; or the material which conducts the vibrations may be more suitable to convey impressions of touch in the one case, and of sound in the other. I wish strongly to insist that you shall give due prominence to investigation by the sense of touch, and not fall into the error of esteeming auscultation as

the be-all and end-all of cardiac diagnosis. I have just had under my care at the London Hospital, two cases in which the existence of thrill, presystolic in rhythm, led me to the diagnosis of mitral stenosis, though, in the one case, there was no murmur at all, and in the other it was so short as to be scarcely distinguishable. You must by no means draw any conclusion from the *absence* of thrill, but where you find a well-marked thrill before the impulse, you have, in my opinion, a certain sign of constriction of the mitral orifice.

We may thus summarise the diagnostic evidence to be obtained from the purring tremor. Felt over (*a*) the base of the heart its rhythm may be (1) systolic or (2) diastolic. If (1) systolic, it indicates (excluding aneurismal thrills, which are outside our subject, and the rare thrill denoting obstruction of the pulmonary artery*) a morbid condition of the aortic valves which has resulted in a narrowing of the outlet from the ventricle. If (2) diastolic, it indicates regurgitation into the left ventricle, owing to imperfect aortic valves. Felt over (*b*) the apex of the heart, the thril may be (1) systolic, or (2) presystolic, but is never diastolic. Systolic thrill indicates regurgitation from the left ventricle through an imperfect mitral valve. Presystolic thrill indicates obstruction afforded by a narrowed mitral orifice to the current of blood issuing from the left auricle.

We will conclude the subject of palpation by a brief retrospect of the evidence which we have obtained. Excluding negative and doubtful evidence, you will observe that the sense of touch has given us

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* *Vide* p. 132.

signs of great and positive value in the following morbid conditions — atheromatous changes in the arterial vessels—hypertrophy of the left or the right ventricle; dilatation of either of the ventricles; pericarditis, in which disease it may have afforded an evidence as to the stage of the disease, whether there is liquid effusion, whether the pericardium is roughened, or whether old disease has resulted in adhesions. Concerning valvular diseases of the heart and their effects, we may have received evidence of aortic obstruction from thrill; of aortic regurgitation from the locomotive pulsation of arteries; of mitral obstruction from presystolic thrill, as well as from pulsation of the left auricle, possibly of mitral regurgitation from systolic thrill; of tricuspid regurgitation from hepatic pulsation.

We shall next consider the evidence to be derived from percussion.

LECTURE IV.

PERCUSSION.

Increased resonance over heart—Increased dulness—Relations of areas of partial and complete dulness—Pericardial effusion—Cardiac hypertrophy and dilatation—Differential diagnosis of the two conditions.

THE object of percussion is to determine the size and shape of the heart and its relation to the neighbouring structures. In my own opinion the best plan of percussing the heart region is to use the fingers only. Place the fore and middle fingers of the left hand *closely* upon the surface of the chest, and tap with a short, decided, but not violent stroke, and in no hurried manner, with the ends of the fore and middle fingers of the right hand. I consider that the fingers of the left hand constitute the best *plexímeter*, and the fingers of the right the best *plessor*. Thus we obtain at the same time information by two distinct routes: the ear obtains evidence of *sound* at the moment that the tactile apparatus receives impressions of *vibration*.

Proceeding by the method of exclusion we will suppose that:

(*a.*) *The præcordial area is resonant;* that is to say, the percussion sound is clear and the vibration unimpaired—there is no dulness. The most common cause of this condition is emphysema of the lung.

The morbidly dilated pulmonary air-cells encroach over the area which, under ordinary circumstances, is occupied by the walls of the heart. Moreover, the heart may be pushed downwards by the too bulky lung. You may have been able, as I have indicated under "Palpation," to feel the beating of the right ventricle at the epigastrium.

A far less common cause of resonance over the heart-region is the presence of air in the cavity of the left pleura (pneumo-thorax). The tympanitic resonance in this condition exists, of course, over the whole of the affected side of the thorax.

In extremely rare cases tympanitic resonance has been found only over the situation of the heart and pericardium; it indicates the presence of air or gas in the pericardium (pneumo-pericardium), a condition which may be induced by fistulous communication with the lung, the œsophagus, or the stomach, or by the decomposition of effused products of inflammation within the pericardial sac.

We will now suppose that:

(*b.*) *The præcordial area is dull.*—We shall have to exclude, first, cases wherein the dulness is not to be distinguished from that existing over the contiguous thorax. Pleuritic effusion in, or empyema of, the left side causes such dulness that the area of the heart cannot be mapped out by percussion. If such liquid effusion be in considerable amount the heart may be pushed completely to the right side, so that its apex may be felt to beat under the right, instead of under the left nipple. Condensations of the left lung, cancer, and abnormal growths in the thoracic cavity also may make it impossible for us to determine by percussion the outline of the heart. Assuming that

these interfering circumstances have been eliminated, we have now to consider the modifications of the percussion-note induced by the heart itself.

We have already considered the normal topography of the heart. We have now to remember that only a certain portion of the heart, covered by its pericardium, approaches close to the thoracic wall—a considerable part being overlapped by the borders of the lung. The portion of the heart which is uncovered by lung in conditions of health, may be demonstrated with sufficient precision in the following manner:—Draw a vertical line through the centre of the sternum. Mark a point, A, on this midsternal line at the level of the fourth left costal articulation. Note the point, B, where the apex of the heart is felt to beat. Join A and B by an oblique line. Complete the right-angled triangle by drawing a line from the heart-apex, B, to the midsternal line at a point, C, just above the ensiform cartilage, at the lower part of the sixth costal articulation. The area enclosed by this triangle will be the portion of heart which, in conditions of health, is uncovered by lung. You should compare it with the area on the thoracic wall occupied by the whole heart as we have before described.

Now to percuss the heart-region. Begin by adapting the two fingers of your left hand held vertically, nails upwards (we are, of course, still supposing our patient to be in the vertical position, sitting or standing), to the thoracic surface, a little to the right of the right border of the sternum. After percussing in this situation, advance the fingers nearer to the midsternal line until the elicited sound is *dull*. Mark the vertical line where dulness com-

mences by a soft pencil or by ink. In the next place commence to determine the left border of the dull area by percussing outside the point of the apex-beat. It is more convenient now to incline the fingers which are adapted to the chest-wall obliquely, pointing towards the sternum, as the left line of dulness will be oblique. Mark the line as previously. The upper limit of the dull area is determined by percussing from above downwards, the fingers adapted to the chest-wall being held horizontally. You have now obtained the upper and the two lateral limits. The lower limit is not so easily determined, because the cardiac merges into the hepatic dulness. The heart and pericardium are close to the left lobe of the liver, separated only by the diaphragm, and I hold that the dulness over these two organs cannot be discriminated. A line of dulness, however, can be determined between the apex on the left, and the commencement of liver-dulness on the right, and you should join these points by a line. For all practical purposes this procedure will indicate to you the area of complete dulness over the heart-region, the *superficial cardiac region* as it has been called, and it demonstrates *the portion of the heart which is uncovered by lung.*

But this is not all that is necessary. You should now reverse the order of your procedure, and starting from the line of dulness, make percussion farther and farther outwards until the sound has the perfectly clear character which it possesses over healthy lung. I do not think it necessary to overburden you with acoustic terms. To understand all that has been written about percussion—sonoriety, pitch, clang, *timbre*, intensity, tones, overtones, fundamentals,

harmonics, &c.—one ought to be able at any moment to conduct an orchestra, tune a harp, read music at sight, and be equally versed in acoustics and practical medicine. Some observers have made assertions as regards the diagnostic power of percussion, that others cannot help thinking extravagant; thus, it has been said that it is possible to ascertain from the percussion-note alone whether a heart has undergone fatty degeneration. It used to be told of Piorry, when I attended his class, that he was able, by knocking at the front door to find out who was in the drawing-room, but such a critical ear is not given to everybody. Suffice it that the sound elicited by percussion over the heart in the situations where the lung overlaps it, is modified by two causes—first, by the nearness of a dense body (the heart) to the point percussed, and, secondly, by the existence between the point percussed and this dense body of an air-containing structure, the lung—*i.e.*, an arrangement capable of transmitting sonorous vibrations. As one proceeds outwards the sound becomes less muffled, and the vibration more manifest, but there is no *abrupt* line of demarcation to indicate the outline of the heart. Nevertheless, you should learn carefully to note where the sound and vibration are uncomplicated (that is where there is lung vibration only), and where they commence to be modified. In estimating the curves of this area, I think it is best to employ as your pleximeter the little finger of the left hand, placed laterally against the thorax, instead of the fore and middle fingers. You should outline the area with ink or pencil; you will now have two concentric figures, the internal being the superficial cardiac area which we have before noted, the external

the so-called "deep cardiac area" corresponding, if it be carefully, and, I think I may add, fortunately, ascertained, to the actual outline of the heart. For practical purposes I would advise you to realize these two areas as, (1) *the area of dulness*, (2) *the area of deficient resonance*, and it is the mutual relations of these that you will find of diagnostic importance. The breadth of (1) the area of dulness in health and in adult life scarcely exceeds three inches transversely. As regards (2), the sound in percussing from above downwards begins to be impaired about the third rib; a curved outline can be mapped out at this level towards the right of the sternum, which indicates the aorta, but you cannot separate the aorta from the heart by percussion.

We will now assume that:

(*c.*) *The præcordial dulness is extended upwards, and its outline is of pyramidal or pyriform shape.* Whenever you find that dulness extends upwards above the third rib, you have strong presumptive evidence of effusion into the pericardial sac. If the sac is distended with fluid you will find that the area of dulness extends from the articulation of the first or second costal cartilage, above to the sixth rib, or sixth intercostal space below.

Here let me insist on the great value of charts or diagrams as records of the physical signs of heart diseases. These may be roughly drawn in a few minutes, for sternum, clavicle, and ribs constitute almost all the outlines that it is necessary to depict. Be careful always to number the ribs as you outline them. Blank chest-charts have been prepared by many observers, and are extremely useful. Dr. Lionel Beale has published some excellent forms

(Churchill); and my friend and colleague, Dr. Stephen Mackenzie, has designed some which I have found very valuable. Inspection and palpation have already indicated points which should be recorded on the chart. The area of visible impulse should be outlined, the position of the apex-beat and the locality of thrill or tactile friction, if these exist, should be marked. Chalks, or pencils of different colours, may be used with advantage. Now you will proceed to record the area of præcordial dulness. If this area be pyriform or pyramidal in shape, with apex upwards, the probability is strong that the case is one of pericarditis with effusion. We will consider the signs that are confirmatory of this view.

In the first place, the transition from dulness to lung resonance is *abrupt*. As I have said, the normal area of cardiac dulness widens gradually into pulmonary resonance. The same occurs in many conditions of disease, but where you have the pericardial sac filled with fluid, the edge of lung, which normally overlaps the cardiac area, is pushed aside, and the area of deficient resonance (as opposed to dulness) is done away with. The line between pulmonary resonance and præcordial dulness is well defined, moreover the sense of resistance to the fingers on percussion is increased; vibrations are lessened.

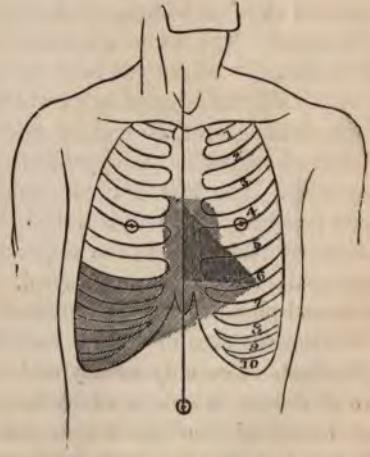
In the second place, there may be noted a peculiar relation between the area of dulness and the position of the apex-beat. You may readily understand that if the area of dulness which you have mapped out be due not to liquid effusion but to enlargement of the heart, you will feel the apex-beat at the lowest limit of the dull area. Not so, however, when there is effusion into the sac of the pericardium. Then the

apex-beat is tilted or floated upwards and you get a certain breadth of dulness between the apex above and the stomach resonance below.

In the third place, you may observe that the extent of dulness varies within comparatively short periods of time. Effusion into the pericardium often takes place rapidly, and you may find that the dull area which you have mapped out previously has, in a few hours, very considerably increased. From day to day there may be advances or recessions of dulness.

The foregoing points may, perhaps, be better understood by reference to the accompanying diagrams, for drawing which I have to thank Dr. Stephen Mackenzie.

Fig. 1.



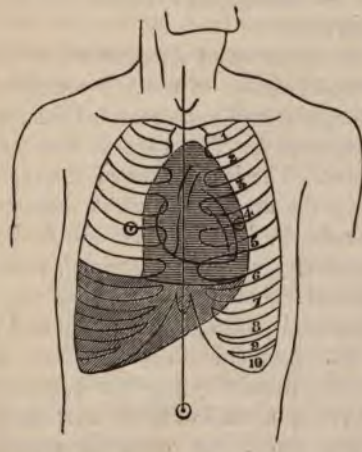
Showing the triangular area of normal cardiac dulness and its relation to the hepatic dulness. The shading indicates the area of deficient resonance—*i.e.*, the deep cardiac region.

Suppose now that:

(*d.*) *The præcordial areas of dulness and deficient resonance are extended laterally.* The impairment of resonance may be extended in either lateral direction.

Assume (1) that it continues to the *left* of the border of the normal triangle of cardiac dulness depicted in

FIG. 2.



Showing the pyramidal outline of dulness due to the effusion into the pericardium, and the relation of the heart to the distended sac.

Fig. 1. This extension of dulness must be due either to hypertrophy or to dilatation, or to both combined, of the left ventricle. In hypertrophy you will have already found that the apex beats below and perhaps outside its normal position. The area of dulness will be sharply defined, but it is only the extreme left of it

which will be much changed in outline. The base line will be prolonged to the left of a line vertically drawn from the nipple, and it will incline downwards and towards the left instead of preserving its horizontal position.

In hypertrophy of the left ventricle you will note that the præcordial dulness preserves its triangular form, but is prolonged towards the left side. You will of course notice the expression of power in the ventricular contraction, as evidenced to the eye, the hand, and the ear.

When the extension of dulness left of the normal area is due to dilatation of the left ventricle, the outline is less angular and more rounded than it is in the case of hypertrophy. The apex is less pointed and more globular. The left border of the region also is less defined; the area of deficient resonance is extended towards the left, and you will find the apex-beat less powerful and less sustained, with the character rather of a short and feeble slap.

As I have said before, hypertrophy and dilatation often co-exist. As a rule, the more obtuse the outline of the apex as obtained by percussion, the greater the probability of dilatation, but the further discrimination between these conditions we shall consider under "Auscultation."

We will suppose now the case that (2) the præcordial deficiency of resonance extends to the *right* of the normal area. We have not, in this case, to determine between hypertrophy and dilatation of the right chambers of the heart, because practically the two conditions always occur together.

You may (*a*) have no difficulty in discovering such *right side hypertrophy*. You may find that the

area of absolute dulness extends beyond the right border of the sternum over the cartilages of the third, fourth, and fifth ribs, and when the right auricle is considerably dilated, you may find dulness extend from the second rib to the third intercostal space. In this case the enlarged heart has displaced the lung which under other conditions overlaps it.

Often, however, (*b*) the determination by percussion of the outline of the right chambers is not easy. Sometimes percussion over the præcordial area elicits no dulness at all. In such case the heart is overlapped by emphysematous lung, and it is precisely in emphysema that dilatation of the right chambers is apt to occur. Disorders of the pulmonary circulation, involving as they do turgescence of the general venous system, lead up to dilatation of the right (the venous) chambers of the heart. Out of forty-five such cases of secondary dilatation of the right heart, Lancereaux observed twenty-four due to emphysema and chronic bronchitis, and six due to respiratory trouble induced by a malformation of the chest through rickets. Though the area of *absolute* dulness may be abolished in such cases, percussion, nevertheless, affords valuable data. You will be able to map out an area of *deficient resonance.* Commencing well to the right of the sternum, where the sound over the emphysematous lung is uncomplicated, you will advance nearer to the middle line of the sternum until you find that the sound, though not dull, is impaired. Thus you may obtain the outline of the deep cardiac area, and closely approximate to that of the heart, covered as it is by lung. You will, of course, observe the concurrent signs—pulsation of the right ventricle at the epigastrium, venous turgescence, perhaps venous pulse, and

if the symptoms are advanced, œdema spreading from the feet upwards.

We have now considered the evidence afforded by percussion of dilatation and hypertrophy of the left and right chambers respectively. You must, of course, be aware that the condition of enlargement may affect both sides. In fact, dilatation of the right chambers may follow from impairment of the propulsive force of the left ventricle as consequence from cause. We shall see this especially when we come to consider valvular incompetence of the left heart. Suppose the left ventricle to be dilated and enfeebled, it is obvious that the first effect is a deficient propulsion of blood through the aorta—the general circulation is impaired owing to the reduced *vis à tergo*—the next result is engorgement of the venous channels; the right chambers of the heart may be considered as part and parcel of these venous channels—like the rest of the venous system, they are in a state of habitual plethora, so they become distended and enlarged, and their muscle becomes hypertrophied.

Having learnt the chief lessons to be derived from percussion of the præcordial area, we shall in the next lecture proceed to "AUSCULTATION."

LECTURE V.

AUSCULTATION.

PART I.

Immediate and intermediate auscultation—Stethoscopes—Normal heart sounds—Auscultation of voice to determine area occupied by heart—Topography of the valves—Modification of normal heart sounds—Accent—Exalted second sound, aortic and pulmonary—Strong pulmonary with weak aortic second sound—Skoda's sign of mitral lesion—Strong aortic second sound in renal disease—Prolonged first sound—Short loud first sound—Reduplication of sounds—Ineffectual systole.

I DO not think it necessary to call your attention to the importance of Auscultation. In the present day this is fully recognised. I am not sure that there is not a slight tendency towards an opposite danger—I mean a too exclusive reliance upon the signs offered to the sense of hearing. When the suggestion has come that the heart-region shall be examined, I have often observed that the stethoscope has been at once flourished, and the listening over the præcordium finished, the diagnosis has been supposed to be mature. I hope to have convinced you by the preceding lectures that precious aids to diagnosis are neglected if auscultation is alone relied on.

On the other hand, the method of diagnosis cannot be complete *without* auscultation.

Auscultation may be direct and immediate, or indirect and mediate.

(*A.*) *Immediate Auscultation.*—You may obtain some general lessons by applying the ear directly to the heart-region, the chest being covered only by a fold of linen. By this you will learn whether the heart is displaced from its normal situation, and whether the sound of its contraction is strong or feeble. If strong and heaving you have corroborative evidence to add to the signs of *hypertrophy*, which have been deduced from previous examination. If feeble, you must hesitate before concluding that the heart-muscle is weak, for it is very probable that fatty tissue or emphysematous lung may intervene between the heart and your ear, and so occasion the feebleness of sound. If loud and heard over a wide area—a wider area than your examination by percussion of the space occupied by the heart would lead you to suppose—it is very probable that you have to deal only with *functional palpitation.*

(*B.*) *Intermediate Auscultation.*—Immediate auscultation is a useful preliminary; it gives you certain broad general impressions—but intermediate auscultation is absolutely indispensable. You have to differentiate sounds coming from situations very close one to the other. The valves of the heart all lie within a square half-inch of surface; if the tricuspid be excluded, portions of all may be covered by a superficial quarter of an inch. Practically, a sound obvious enough at a given point, may cease to be perceptible one-third of an inch therefrom. It is obvious, therefore, that the unaided ear cannot be relied upon, but that some means must be adopted for collecting and localising sounds. The stethoscope must be used.

A few words as to the form of stethoscope most

suitable. The ordinary wooden stethoscope is the most generally useful; the thoracic end should be small enough to be adaptable to the intercostal spaces in thin subjects. As a rule, I think the metallic stethoscopes are far inferior.

The binaural stethoscope is extremely valuable. For the cardiac auscultation of infants and children, it is indispensable. Not only does it shut off from the ears external sounds and allow the impressions to come only from the part at which its cup is applied, but its flexible collecting tubes allow each movement of the patient to be followed. Otherwise, children and infants may elude all your attempts at auscultation. Moreover, the sounds obtained through the double tube of this stethoscope, transmitted as they are to both ears, are perceived very intensely: sounds may be heard which otherwise would be inaudible. Still, I fully recognise that there is a danger—the sound may be too intense, so that if two occur near together, one may be drowned. The practical conclusion is, *use both forms of stethoscope*, the ordinary and the binaural. I have my own stethoscope so constructed that the same cup and stalk can be screwed into the ordinary wooden ear-piece, or into the junction of the tubes of the binaural arrangement.* So I examine successively with each form of instrument.

Now it is necessary briefly to consider the NORMAL SOUNDS OF THE HEART. The mere application of the ear to the præcordium will convince you that the noise of the heart in action can be resolved into two

* This has been made for me by Messrs. Maw, Son, and Thompson.

sounds, which can be imitated by the syllables, *lubb-dup*. A little consideration will show you that the first, the longer sound of lower pitch, is separated from the shorter, sharper sound by a short but distinctly appreciable interval of silence, and that, again, a longer pause or silence intervenes between the second sound and the recurrence of the first. Listening near the situation of the apex, you will find that the first sound co-exists with the impulse of the heart against the walls of the chest—that is to say, the contraction of the ventricles, the systole.

Without entering in the spirit of criticism upon the much-debated question of the exact mode of causation of these sounds of the heart, we shall find it sufficient for our present purpose to consider what is taking place at the exact time at which these sounds respectively are occurring.

At the time of the production of the *first sound*, the following are the conditions. The ventricles are full of blood. The auricles have just contracted, impelling their contents through the auriculo-ventricular openings, and completing the replenishment of the ventricles. Then the muscle of the ventricular walls contracts—the tension of the enclosed blood forcing upwards the curtains of the auriculo-ventricular valves, putting them suddenly upon the stretch, and thus closing the orifices which they guard—driving the blood contained in the ventricles through the only now pervious openings—viz., the aorta on the left side, and pulmonary artery on the right.

At the time of the production of the *second sound* the conditions are these. The aorta, and through it the arterial channels of the whole body, as well as the *pulmonary* artery and its branches which carry

venous blood to the lungs, have just been filled with a gush of blood. A momentary interval has occurred whilst these onwards currents have passed, and during which the ventricles have commenced to become relaxed. The blood has been forced by the systole not into rigid tubes or mere inert canals, but into the elastic arteries. When, therefore, the ventricles have ceased their contraction, and an appreciable pause has occurred during which the impetus of the blood-current has been unresisted, the semilunar valves of the aorta and pulmonary artery respectively are suddenly closed. The cause of such closure is twofold. First—the mere weight of the superincumbent column of blood in these great vessels; secondly—the elastic recoil of the previously stretched coats of the vessels of distribution. In the case of the aorta, the elastic recoil comes not only from itself, but from every artery and arteriole in the whole system—in the case of the pulmonary artery, from the narrower limits of the distributing channels within the lungs. The second sound, then, is short, sharp, and sudden, aud there is no doubt that it is due to the sudden closure of the semilunar valves of the pulmonary artery and the aorta.

During the pause following the second sound, the heart muscle is flaccid, and the cavities are becoming refilled.

The first sound, then, is coincident with the contraction of the ventricles; it is *systolic*. The second occurs at the moment of closure of the semilunar valves; it is called *diastolic*, but it is obvious that it occupies only a portion of the diastole. The diastole of the heart embraces all that period which is not occupied by systole.

Supposing the period of rhythm to be divided into ten equal parts, the following expresses, according to Dr. Walshe, the relative duration of sounds and silences:

First sound	=	·4
First silence	=	·1
Second sound	=	·2
Second silence	=	·3

In commencing the auscultation of the heart's area, you may have a duty as yet unfulfilled. Your former investigation may have failed to have indicated the situation of the heart's apex. This you must now determine by means of the stethoscope; observe and mark the extreme left of the situation at which the impulse is heard at its maximum; that will correspond to the apex.

It may be necessary also to employ auscultation as a corroborative means of determining the area occupied by the heart, or by distended pericardium. You do this by auscultating the voice. The lines where vocal resonance terminates or becomes greatly diminished, indicate the border of the area.

We have now considered the necessary preliminaries for Auscultation of the Heart. The apex has been indicated, and the area occupied by the various chambers has been mapped out. We have already described the topography of the heart; we have now to consider the TOPOGRAPHY OF THE VALVES. The aortic, pulmonary, and tricuspid valves all are situated near the surface of thoracic wall—not so the mitral—this only approaches the surface at the point of the apex-beat, where only the left ventricle comes towards the thoracic surface.

The relation of the situations of the various valves to the sounds which they occasion has been so clearly expressed by Dr. G. W. Balfour, that I cannot do better than quote his words:—"We find that sounds produced in any one cavity of the heart are heard with most distinctness over that part of the thoracic wall at which the given cavity approaches the surface most closely; thus the only point at which the left ventricle directly impinges on the chest-wall is where the apex-beat is felt, and that is precisely the spot where the first sound, produced in the left heart by the closure of the mitral valve, is most distinctly heard: *a space of about an inch in diameter around the apex-beat is therefore termed the mitral area.* Nearly the whole of the right ventricle is uncovered by lung, and impinges directly on the lower part of the sternum; and at this part, especially along the left edge of the sternum where it is joined by the cartilages of the fourth, fifth, and sixth ribs, the right first sound produced by the closure of the tricuspid valve is best to be heard, and *the triangular space covering the position of the right ventricle is therefore termed the tricuspid area.* . . The aorta and pulmonary artery originate very close to each other, the pulmonary valve lying about the middle of the third left cartilage, one half being to the left and the other to the right of the left edge of the sternum, which exactly divides it in two. From its point of origin, the pulmonary artery rises to the lower edge of the second left cartilage, where it divides into its two great branches going to the right and left lungs; this, therefore, is the place where it is nearest the surface of the chest, and *the second left interspace or the sternal end of the third cartilage, is the position in which*

the pulmonary second sound is best heard. On the other hand, the aorta rises a little below, behind and to the right of the pulmonary artery, its valves corresponding to the lower edge of the third left cartilage, behind and to the right of the pulmonary valves, and it passes upward, forward, and to the right, till it reaches the upper border of the second right cartilage, when it passes obliquely backwards and to the left, forming what is termed the transverse portion of the arch of the aorta. *At the second right cartilage, therefore, the aorta is nearest the surface of the chest,* and the arterial walls and the blood-current coincide in readily conveying in this direction the resonant vibration, which results from the closure of the aortic semilunar valves; in this position, therefore, the aortic second sound is most readily differentiated from the pulmonary one."*

Deferring for a time any consideration of abnormal sounds heard over the heart's area, we will consider:

(*a.*) THAT THE HEART-SOUNDS ARE MODIFIED IN DEGREE.

A few words as regards *accent.* If you listen over the apex of the heart you will find that the more pronounced of the two sounds is the first or systolic one. The accent falls on the sound produced by the contraction of the ventricles, such sound more readily reaching the ear. It would be expressed by the syllables, *lubb-dup.* At the base, however, the accent

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* *Vide* A Lecture on the Diagnosis of Disease of the Heart, by George W. Balfour, M.D., F.R.C.P.E., Physician to the Royal Infirmary, Edinburgh. Reprinted from the *Edinburgh Medical Journal* for June, 1874. [Edinburgh, Maclachlan and Stewart; London, Hardwicke.]

is reversed. The semilunar valves are nearer to the ear, the accent falls on the shorter second sound and the expression would be *dup-lubb*. This may give rise in some cases to a little perplexity: you may take the second sound for the first and *vice versâ*. The difficulties may be obviated, however, by a very simple rule. When you are auscultating the base of the heart, place the finger over the spot of the apex-beat, or, if this be too feeble to be distinguished, over the situation of the great vessels at the root of the neck. The impulse or the pulse will thus tell you which is the first sound: of course if the sound you hear be synchronous with the systole or pulse it is the first sound, if not it is the second.

It is well, in my opinion, to commence by auscultating at the base of the heart, because the situations of the aorta and pulmonary valves are fixed points, whilst the apex is variable.

At the base, in the absence of abnormal sounds, it is to the *second* sound that you must pay attention. The quality of the first sound will be investigated at the apex.

You will place your stethoscope over the second right costo-sternal articulation, then carry it across the sternum to the second left interspace and the articulation of the sternum with the third costal cartilage on the left side. You will observe and compare the qualities of the second sound in these right and left situations respectively. The aortic second sound is normally more pronounced than the pulmonary. It is the closure of the aortic valves which gives, for the most part, the character to the second sound which is heard over the general area of the heart—that is to say, the aortic overpowers the pul-

monary second sound. Suppose now that (*a*) *the aortic second sound is intensified.* It is obvious that you must train your ear by observing the second sound in cases of health. When you have recognised its normal intensity only are you in a position to judge of its exaggeration. If on placing your stethoscope over the second right costo-sternal articulation you hear a sharp and loud flap coming closely to the ear your inference must be that an abnormal amount of blood, which has been forced into the aorta by the contraction of the left ventricle, has, by the energy of its reflux, closed and put on the stretch the semilunar valves. Carrying your inference further, you will find that you must have a strong, muscular, in other words, *hypertrophied left ventricle*, and an aorta capable of containing an unusual amount of blood, in other words, a *dilated aorta.* In conjunction, therefore, with other signs you will find persisted exaggeration of the aorta second sound a valuable evidence of hypertrophy of the left ventricle. Conversely, if you find that (*b*) *the aortic second sound is feeble*, you may infer that there is a deficiency of arterial blood supplied to the aorta by the ventricle. This observation is sometimes valuable as a means of prognosis when the blood has been diminished in quantity by hæmorrhage, or when the tone of the heart has been enfeebled by disease. In fevers a very weak second sound is a bad prognostic. There are certain circumstances, however, which mar or modify such absolute deductions from the observed force or feebleness of the second sound. There may be a sharp and loud second sound when the aortic valves are unusually thin. Or it may have a dull or leathery character when the valves are thickened. You must, therefore,

remember to record the observation, but to make no absolute deduction without comparison with other physical signs.

Suppose now that (*e*) *the pulmonary second sound is intensified.* This is a sign of more absolute importance. I have said that the great cause of intensification of the aortic second sound is an increase above the normal quantity of blood in the aorta. In case of the pulmonary second sound another cause is present to augment it. The pulmonary is a smaller circuit, and the blood contained in it, like the water in a Bramah press, exerts equal pressure in all directions. The blood-pressure in the general systolic circulation is modified in various ways, by subsidiary circulations, as the portal, and by many special conditions in the various tissues. The pulmonary circulation may be looked on as if conducted by a single flexible and contractile tube from right ventricle to left auricle. If, from any cause, therefore, there is any obstruction to the flow of blood within the pulmonary circuit the result is not only congestion of the lungs, but also "a uniform increase of the tension throughout the whole of the pulmonary circulation, often accompanied, if long continued, by slight dilatation of the pulmonary artery, and always by a closure of the semilunar valves with an exaggerated force proportionate to the hindrance it has met with."*

Of still greater importance than the estimation of the absolute strength or weakness of the aortic and pulmonic second sound is the observation of the relative force of the sound in the two situations.

Suppose that you find that (*d*) *the pulmonary second*

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* Balfour, *loc. cit.*

sound is intensified whilst the aortic is enfeebled. Your first inference will be, as I have just indicated, that there is exaggerated tension in the pulmonic circulation—that is the cause which must produce the exalted second sound. This may occur, however, in any condition of respiratory trouble when there is congestion of the lungs, but then the second sound over the aortic valves need not be perceptibly or persistently enfeebled. The co-existence of a strong pulmonary with a weak aortic second sound is a sign that whilst the pulmonary artery receives too much blood the aorta receives too little. It is valuable evidence, as Skoda first pointed out, of a diseased condition of the mitral orifice either permitting regurgitation, or inducing obstruction of the blood-stream. In the case of the former the explanation of the phenomenon is easy: the arterial current, urged into the aorta by the contraction of the left ventricle, is diminished by the amount which regurgitates through the imperfectly closed mitral orifice; the aorta, therefore, is insufficiently supplied with blood, and the sound caused by the reflux against its semilunar valves is consequently weak. In the case of mitral obstruction (stenosis) the effect is the same, though the cause is different. Here the blood reaches the ventricle with difficulty on account of the narrowing of the outlet from the auricle, systole takes place upon an insufficient amount, and the aorta is ill supplied. The case is just otherwise with regard to the pulmonary artery; as a consequence of the deficient arterial supply, induced by both the above conditions, there has been a reduction of the rate of venous return, an engorgement of the venous system, a secondary dilatation of the right side of the heart. In regurgitation a back*ward current* is created by the systole of the ventricle;

in obstruction the contraction of the auricle tends to cause reflux. All these causes, therefore, produce high tension in the pulmonary circuit. So, with the arterial anæmia there is venous hyperæmia, and hence exaltation of the shock of recoil of the semilunar valves pertaining to the right heart. This observation of the comparative intensification of the pulmonary second sound is valuable, not only as a sign of the existence of the conditions I have just mentioned, but as an indication of their degree—so it is of great importance in prognosis. In proportion to the comparative feebleness of the aortic second sound is the failure of the ventricle; in proportion to its strength is the power of the ventricle, by its compensatory hypertrophy, to overcome the obstacles imposed.

If you find the opposite condition, when (*e*) *the aortic second sound is relatively intensified*, it is evidence, of course, of hyperæmia on the arterial side of the circulation. I have already spoken of this as suggesting hypertrophy of the left ventricle, but it is only to be relied on in concurrence with other signs. There may be much hypertrophy of left ventricle concurrent with mitral regurgitation, when, as we have just seen, the aortic second sound is enfeebled. In such case the hypertrophy is insufficient to compensate for the loss by regurgitation through the mitral orifice. Again, though the left ventricle may be hypertrophied and the aorta dilated, the coats of the latter (through atheroma for instance) may be so inelastic that the second sound may suffer no exaltation. There is one condition of disease, however, in which accentuation of the aortic second sound is a sign of considerable importance—this is *Chronic Bright's Disease.* I have called your attention already to the conditions which exist in this disorder. There is a peripheral obstruc-

tion to the circulation of the blood through the terminal arterioles, coincidently there is heightened power of left ventricle, the necessary consequence is an abnormal excess of tension in the arterial system, and this state of tension occasions the sharp and pronounced aortic second sound.

We turn now from the base to the apex; and our consideration will be devoted to the first instead of the second sound. Suppose we find that (*f*) *the first sound is abnormally prolonged.* We have then evidence of hypertrophy of the left ventricle. In auscultating at the apex I advise you to keep distinct in your minds the element of *sound* and the element of *duration.* You see that as evidence of hypertrophy I have called especial attention to the latter. The first sound is for the most part constituted by two factors, the contraction of the ventricular walls, and the tension of the auriculo-ventricular valves. The muscular sound of the contracting ventricles is dull and prolonged, whilst the sound of the stretching of the curtains of the valve is (as you know it to be in the case of the semilunar valves) sharp, short, and sudden. In proportion, therefore, as the muscular element of the first sound preponderates over the valvular, so the first sound will be dull and prolonged. If, on the other hand, (*g*) *the first sound is shortened*, it is evidence that the ventricle is weak. In this relation we may have several conditions. The first sound, though short, may be loud. It is rather a frequent mistake to interpret loudness of the first sound as a sign of increased power of the ventricle; as a rule it is the opposite. A loud, sharp, short, flapping first sound at the apex indicates dilatation, and consequent feebleness of the ventricular walls. In such case the *muscular* sound is reduced to a minimum, and the ear

appreciates in the greatest degree the valvular element of the first sound. Then the first sound approaches the second sound in character and duration. You may even be puzzled to discern the one from the other. The following rule will easily guide you: whilst you are listening by means of your stethoscope, place your finger over the spot nearest the heart apex, at which a decided pulsation is to be felt. If the apex-beat is evident, place your finger over its situation, if not, observe the carotid pulse (not the radial, for that is fallacious, because it is not always coincident with the cardiac systole). You thus have a certain indication of the time of the systole, and you will be able at once to know whether the sound which you hear is synchronous with it—*i.e.*, the first sound—or intermediate between pulse and pulse, when it will, of course, be the second sound. But the first sound may be short and yet feeble, and this is evidence of a still graver condition of heart-debility. You then have, not only an impaired muscular sound, but a weak valve sound, for the enfeebled muscle cannot create sufficient tension to produce the pronounced flap of the valvular curtains. In such case you must look for the other signs and symptoms of degeneration of the muscular walls of the heart. In extreme cases of fatty degeneration, the first sound may be lost altogether.

We will next consider briefly a condition which is often more interesting than important, but which must always be noted and deciphered where it exists—(*h*) *the sounds of the heart are reduplicate.* Instead of the single sound of the normal rhythm you hear two sounds following as if the syllables *tah-ta* were uttered. This may occur at the base or at the apex, or in both situations. It may be heard as a per-

manent sign in conditions of health, as a transient occurrence in such conditions, as a permanent sign in disease, and lastly, as a phenomenon, continuing definitely during a morbid condition of the heart, and vanishing when health becomes restored. On what does it depend? The inquiry is interesting, but the answer is not easy. We will first take the case in which the phenomenon is manifest at the apex and not at the base, though this is comparatively a rare condition. Now I must ask you carefully to discriminate between *reduplication* and *ineffectual systole*. I have already said that in some cases contraction of the ventricles may be repeated without producing a pulse to be felt at the wrist.

Many cases and observations have been recorded which illustrate this ineffectual working of the ventricle, notably by Von Dusch, Potain, Hayem, and Flint. This is not reduplication, however; in reduplication there is a repetition only of a portion (the systolic or diastolic sound as the case may be) of the cardiac revolution, whilst in ineffectual systole the whole cardiac revolution is repeated once, twice, thrice, or even four times, until sufficient arterial tension is attained to produce the pulse. In such case the pulse may be sometimes felt in the larger vessels, as the carotids, before it is manifest in the radials. You may, perhaps, understand this better if I indicate it graphically. Let the syllables *tah-ta* express the cardiac sounds, *tah* being the first sound and *ta* the second. Then INEFFECTUAL SYSTOLE is thus represented:—

tah-ta: *tah-ta* = Pulse: or
tah-ta: *tah-ta*: *tah-ta* = Pulse: or
tah-ta: *tah-ta*: *tah-ta* TAH-TA = Pulse,

the latter indicating varying expressions of strength of the sounds.

REDUPLICATION OF THE FIRST SOUND is represented by

tah-tah : *ta* = Pulse : tah tah : ta = Pulse, or
tah *tah* : ta = Pulse : tah *tah* : ta = Pulse.

The first idea occurring to you as an explanation of this doubling of the first sound may be that there is a want of unison in the contraction of the ventricles, one ventricle delaying its contraction. This explanation, however, it is very difficult to accept, when we consider the structural unity of the ventricles, and the observed consentaneousness of their action. It is not so difficult to realize that, although their contraction commences at the same moment, complete systole, owing to different conditions of blood-pressure and other causes which we shall presently consider, may be accomplished in one before the other. This is probably the true explanation. A doubling of the first sound may be heard in health at that period of the respiratory rhythm when expiration has just been completed and inspiration is commencing—that is the period of greatest blood-pressure in the eight cavities—and the reduplication is caused by retarded closure of the tricuspid valve.

We will now consider reduplication of the second sound, which is much more commonly observed. This may be expressed by

tah : *ta ta* = Pulse : *tah* : *ta ta* = Pulse, &c.

There is but little doubt that this is caused by a want of a unison in the closure of the semilunar valves of the aorta, and the pulmonary artery respectively. If there be disproportionate blood-pressure

in either of these vessels, there will be a tendency on the part of the valves pertaining to such vessel to a more early closure. Potain noted that reduplication of the second sound occurred in health at the end of inspiration and beginning of expiration, when there is heightened blood-pressure in the aorta. Exaggeration of tension, however, in either of the vessels is not, in my opinion, the only explanation of the phenomenon. I consider that a doubling of the second sound is often produced by the same cause which occasions doubling of the first—namely, an arrest of the contraction of one ventricle before the completion of the contraction of the other. As a consequence, one ventricle enters into diastole before the other, and hence, of necessity, reflux against, and closure of the valves of its vessel of exit takes place at an earlier moment. This explains the very rare cases in which doubling of both first and second sounds, a quadrupling of the normal sounds has been observed. Doubling at the apex occurs on occasions when the valve sound of the tricuspid is audible; the more frequent doubling at the base is readily explained by greater aptness of the semilunar valves to produce sound than the auriculo-ventricular.

Doubling of the second sound may take place in conditions of debility of the muscular structure of the heart. It is often observed in the course of typhoid fever, commencing usually in the second week. In such case M. Hayem has shown that there is an inflammation of heart muscle. The reduplication ceases as health is regained.

I have already said that doubling of the heart-sounds may take place even persistently in conditions of health. Then it is probably due, like the other instances of harmless cardiac irregularity, to causes

resident in the nervous system. Much more frequently, however, it is the result of organic disturbance. As regards the second sound, conditions of varying blood-pressure in aorta and pulmonary artery respectively may give rise to it, whilst rhythmical disturbance to the ventricles may occasion a doubling both of first and second sounds. Such rhythmical disturbance may, in my opinion, have its origin in the nervous system, in the muscular wall, or in the conditions of blood-pressure within the ventricles. Any cause which makes the contraction of one ventricle to be completed before that of the other—whether that cause be an over-repletion of one of the cavities, whereby the closure of the auriculo-ventricular valve pertaining to it is retarded, or a morbid condition of the muscular walls of the ventricles (myocarditis or degeneration), whereby the fibres of one ventricle contract more feebly or become more quickly exhausted than the other, or a condition of nervous supply hastening the systole of one of the cavities—can occasion reduplication of the second sound, or of the first sound, or of both.

I have said that these phenomena are often of more interest than importance, but you must on no account pass them over as trivial, or merely note the occurrences without an endeavour to discover the causes. You will meet with many instances of disturbed rhythm of the heart which are exceedingly puzzling, but by attention and repeated examinations you will be able to resolve them in most cases. A tumultuous and irregular succession of sounds, which at first seem chaotic, may frequently be analysed, and found due to a definite association of morbid and normal heart-sounds.

LECTURE VI.

AUSCULTATION.

PART II.

Abnormal sounds—Pericardial friction-sound—Relations between pericarditis, rheumatism, and renal disease—Influence of posture on friction-sound—Exocardial friction—First-sound murmur over aortic valves—Anæmic murmurs—Murmurs in chorea—Aortic stenosis—Ulceration of aortic valve-segments—Aortic second-sound murmur—Evidence from ophthalmoscopic examination—Aortic insufficiency—Conduction and convection of murmurs—Double aortic murmur.

HAVING considered the modifications of the normal sounds we will now turn to our second subdivision and assume :—

(*b.*) THAT ABNORMAL SOUNDS ARE HEARD OVER THE HEART REGION.

Abnormal sounds, heard at, or close to, the situation of the heart, accompanying the cardiac movements and apparently mingling with some of the normal sounds, may have their origin (1) in the structures external to the heart and pericardium; (2) in the pericardial sac; (3) in the muscular tissues of the heart; (4) in the endocardium and the valves of the heart; (5) in the blood transmitted by the heart.

As regards the first-mentioned cause of abnormal sound, it is of course assumed that you have carefully estimated the pulmonary conditions, especially of the

left cavity of the thorax and the lung which borders on the heart region. The diseases which you have chiefly to consider as inducing sounds which resemble those due to intrinsic diseases of the heart, or as modifying and irregularly conducting the sounds due to intrinsic diseases, are pleurisy with viscid effusion, cavities in the pulmonary substance near the heart, and condensation of the lung. The differential diagnosis of these conditions we shall consider as occasion arises.

We will assume that:

(1.) A rubbing or creaking sound, accompanying both movements of the heart, is heard superficially over the cardiac region, but is not intensified at apex or base.

This indicates a diseased condition of the pericardium in which the visceral and parietal layers are separated by a viscid or fibrinous effusion, or in which the pericardium itself has become thickened or roughened. Your previous examination may have given the clue to your diagnosis. You may have concluded that the pericardial sac is more or less distended, and the symptoms may have assured you that there is in existence a pyrexial disease accompanied by cardiac, pulmonary, and, perhaps, cerebral phenomena. You may even have had the more positive evidence of pericardial friction fremitus. Moreover, you may have heard the friction-sound by direct application of your ear to the chest before commencing to use your stethoscope. If you suspect pericarditis you should, in my opinion, never omit the method of direct auscultation.

But you may have none of these signs to guide you, and let me insist strongly on this. Your patient may

have pain referred to some of the joints; these pains may be very trivial in character, may, indeed, be absent; pyrexia may be very slight or inappreciable. I mentioned in an early lecture a case of pericarditis in the most pronounced degree which went through its course without a single symptom of distress. I have lately had under my care, at the North-Eastern Hospital for Children, a boy who manifested the disease with its grave progressive course and complications, but with the occurrence of so few subjective symptoms that it was impossible for me to persuade the parents to leave the child as an in-patient, and he was brought to me bi-weekly as an out-patient. In this case there was loud pericardial friction, which, as it faded away, gave place to the murmur of mitral regurgitation, indicating that endocarditis had attacked and spoiled the mitral valve; at the next consultation it was evident that the disease had spread to the aortic valves, for there was a murmur of aortic obstruction, and at the following there was, in addition, a murmur of aortic regurgitation, showing that the endocarditis had rapidly destroyed the integrity of the aortic valves and induced their most dangerous lesion. You will remember, then, that you are not to look for pronounced symptoms to suggest to you the occurrence of pericardial friction.

I have said that your patient may complain of pain in one or more of the joints. This rule you are most strongly to observe—carefully to examine the heart and to suspect pericarditis in any case manifesting acute or subacute rheumatism. By far the most common form of pericarditis is that which is associated with rheumatism. Many circumstances govern the frequency of the manifestation of peri-

carditis in rheumatic fever, and authors vary in their estimate of such liability even from 16 to 37 per cent. I have compared the figures recorded by Fuller, Von Bamberger, Roth, Leudet, Duchek, and Chambers, and I find that they give about 24 per cent. as the ratio of cases of pericarditis occurring in acute rheumatism.* Of 50 cases of recent pericarditis Flint observed that 19 were manifestly rheumatic. Even this high figure, however, does not represent the prevalence of the rheumatic form of pericarditis, for I have already drawn your attention to the fact that pericarditis occurs in the subacute form of rheumatism, and in cases where the articular phenomena are very slightly developed. In some rare cases the process of rheumatic fever commences by pericarditis.

Another condition of disease in which you must recollect that pericarditis is probable is renal disease. In all cases of pericarditis, but especially in cases occurring after adult age, you should examine the urine for albumen. This is highly important from the point of view of prognosis, for whilst rheumatic pericarditis generally ends in recovery, pericarditis occurring with renal disease is generally fatal. The disease may also develop in morbid conditions of the blood as scurvy, and, though rarely, in the course of scarlatina or variola.

Your previous examination will, in most cases, have indicated that the pericardium contains some liquid effusion; but this is not invariable. Peri-

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* The results of the following observers approach very closely and are probably nearest the truth: Fuller, 16·7; Duchek, 16; Chambers, 18 per cent.

carditis, with friction-sound, may occur, when the exudation is inappreciable (Pericarditis sicca), or you may examine the case at a period when most of the effusion has disappeared by absorption.

The importance of the observation of the sound of pericardial friction is very great. This is one of the very few great points that we have gained since the time of Laennec. In most cases it is marvellous to see how close and complete were the observations of this great master. Our advances in physical diagnosis since his day have been neither great nor rapid, but in this case there is an exception. Laennec says:—"There are few diseases more difficult to recognise than pericarditis, or more variable in their symptoms. . . . I must acknowledge that mediate auscultation does not afford much more certain signs of pericarditis than the study of the general and local symptoms." Thanks to the subsequent observations of Stokes, Watson, and Bouillaud, we now know that the existence and course of pericarditis can be traced with the same precision as in the case of other cardiac diseases.

It is very rare indeed that a friction murmur fails to be developed at some time during the evolution of the pericarditis. It appears early in the disease. Sometimes it disappears also early. Walshe has known it appear and disappear within six hours. Sometimes it appears, then disappears for a time during a period of greater abundance of liquid effusion, then reappears, to vanish gradually. Most frequently it lasts for many days, varying in character and intensity, and is heard over a less and less extended area as the disease terminates.

The characteristics of the pericardial friction-sound

are chiefly—(1) Its quality; it is a "rubbing" or "creaking" sound, resembling that produced by the attrition of two surfaces of cloth or leather. It accompanies both movements of the heart, and is admirably indicated by the expression of Sir Thomas Watson—a "to-and-fro" sound. This rule is subject to very rare exception when the rub is heard only with the systole. We must postpone consideration of this till we come to the differential diagnosis of valvular murmurs. (2) Its limitation. It is heard only over the superficial cardiac region. This is very important. The sound is not conveyed in certain directions from the heart-area, as we shall find to be the case in regard to endocardial murmurs. It is situated over the heart-muscle, and is associated more with the *movements* than with the normal *sounds* of the heart. At apex and base these normal sounds may be heard — distant and feeble if there be much effusion. On applying the stethoscope over the intervening area occupied by the heart-muscle (chiefly the right ventricle), the rubbing sound becomes manifest. (3) Its superficiality. It seems to be generated near the ear. If, as the late Dr. Sibson pointed out, you press your stethoscope firmly upon the chest-wall, the sound becomes more developed, and its quality may be altered—it may become rougher. The audibility of the friction may be found to be directly proportionate to the amount of pressure. I need not say that you must be careful not to inflict pain. Again, if you withdraw your ear slightly from the chest, you may still hear the sound. Dr. King Chambers calls attention to the following plan, as affording valuable evidence:—Having observed the sound with the ear, as usual, close to the

stethoscope, gradually withdraw your ear, the stethoscope remaining applied to the chest, the sound of pericardial friction will still, with great probability, be recognisable. This nearness of the sound to the surface is valuable in the differential diagnosis between exocardial and endocardial murmurs.

The friction-sound, however, may be very slight and faint. In such case you should make your patient change posture; the friction may then become more manifest. The intensity may be increased by a change from the vertical to the recumbent position, or *vice versâ*, or by inclining the body backwards or forwards. In some cases a friction murmur is only heard when a particular position is assumed by the patient. When, therefore, your examination has led you to suspect pericarditis, especially when you are satisfied that there is effusion into the pericardial sac, and you are anxious to discover evidence that the exudation is fibrinous and tending to absorption, you should carefully auscultate in various positions, and note the presence or absence of friction-sound.

Attention to the points I have mentioned will generally enable you to recognise the existence of a pericardial friction-sound; but there is one condition in which the diagnosis may be difficult. This is when the friction is produced, not within the pericardial sac, but outside, in the pleura.

You will remember the rule I adduced—to examine carefully for disease in the left side of the chest. Suppose that your examination has led you to conclude that there is pleurisy with effusion, or pneumonia, or that the lung abutting on the heart-region has undergone any of the changes occurring in the

course of pulmonary phthisis. If, in such conditions, you hear a "to-and-fro" sound over the præcordium, remember that this may be a *pleural* friction, modified by the movements of the neighbouring heart, and so made to resemble the pericardial rub. The pleural surface may be locally roughened by recent exudation, or thickened by more remote, or by progressive inflammatory changes. If you have such a sound under such conditions, and there is no other evidence of the existence of pericarditis, the balance of probability will be in favour of its exocardial origin. To further the differential diagnosis, observe whether the sound varies in character, and what are its relations with the rhythm of respiration. Does it vary in intensity in an irregular manner? Does it become imperceptible in some of the cardiac revolutions? Is it more pronounced at the end of a full inspiration? Observation of these points will help you to a correct conclusion.

A pericardial friction-sound is less liable to variation than a pleuro-pericardial: the latter is most evident when the lung is inflated, and thus the rough pleural surface more closely adapted to the pericardium, whilst the pericardial sound is most pronounced when the heart is least covered by lung, that is to say, at the end of expiration.

Having excluded the sounds which take their origin in the structures superficial to the heart, we come now to a wide field of inquiry—that which includes the morbid sounds intrinsic to a diseased heart. The subdivision of abnormal sounds which we now consider is that wherein:

(2.) Abnormal sounds, occurring with or replacing the normal sounds are heard only, or with a maximum

intensity, over the various situations of the valves, or in definite relation with such situations.

You have applied your stethoscope over the aortic cartilage (the point where the second right costal cartilage joins the sternum), and you hear a soft blowing sound; placing the tip of your finger over the apex of the heart, or over the carotid artery, you are convinced that the murmur coincides in time with the pulse—that is to say, with the first sound of the heart. Moving your stethoscope so as to auscultate in a direction upwards beneath the right clavicle or downwards over the heart, you find that the sound is lost. It is, in fact, *a first-sound murmur localised over the situation of the aortic valves*, and it indicates an alteration of the segments of these valves or a change in the normal aortic orifice: the form and nature of such change we shall consider presently.

Suppose, however, that you are satisfied that *whilst the sound which I have just mentioned is not propagated downwards towards the heart, it is conveyed in a line extending towards the right clavicle.* This may possibly be due to an *aneurism* of the ascending part of the arch of the aorta, or of the innominate artery. To establish or eliminate this hypothesis, you must examine carefully for the concurrent signs of such condition—a local prominence pulsatory to the touch and perhaps communicating a thrill, a localised area of dulness over the affected vessel, signs of alteration of circulation in the distal branches of the diseased vessel, and effects of the progressive pressure on neighbouring structures, caused by the growth of the aneurismal tumour.

You must remember, however, that whilst the occurrence of aneurism may explain the existence of

the murmur, the hearing of this murmur is by no means necessary to establish the existence of aneurism. A systolic murmur is absent in a large number of cases of aneurism.

Aneurism eliminated, you have next to consider whether the murmur may be *anæmic*. Here let me say that you may find the differential diagnosis not at all easy. Carrying your stethoscope in the direction of the right subclavian artery, the right carotid, the left subclavian and the left carotid, note the points where the murmur disappears, and, if this happens, where it reappears. In the great majority of cases an anæmic murmur is a soft murmur; it is heard in the course of the great arterial vessels, not with a diminishing intensity, as one recedes from the heart, but often with reinforcement over the arteries; a slight increase of pressure made by the stethoscope increases the loudness of the murmur or develops it when it is not heard. There is one observation which will give you positive aid in the differentiation. Having caused your patient to turn the head towards the left, apply your stethoscope above the right clavicle in the hollow behind the sterno-cleido-mastoid muscle: you may now hear a continuous musical hum, the origin of which is in the great veins. You can at once distinguish it from the murmur which you have just heard in the arteries, because, whilst the latter is systolic, occurring only with the arterial pulse, the former is an uninterrupted sound, a sound called by French observers the "bruit de diable," or "humming-top sound." By making pressure with the finger over the veins above the stethoscope, the murmur will be made to cease—a sufficient proof of its venous origin. You should

auscultate in like manner on the left side of the neck. If you hear the venous hum in either or both of these situations you have strong evidence that the murmur which you have heard at the base and up the vessels is a so-called hæmic or anæmic murmur. But you obtain very valuable collateral evidence from the general condition of your patient. If there are the pallor and usual signs of anæmia; if your patient is a female of an age when the catamenia are commencing, or at a later age, when there are troubles due to excess or deficiency of the menstrual function, or to leucorrhœa, or at a later age, when there has been much loss of blood—or if the case is one of a male, who has suffered from hæmorrhage or some potent debilitating cause, or presents the signs of early phthisis, the probability of the murmur being anæmic is rendered very great.* I have seen it stated that anæmic murmurs are seldom or never met with in young children. I can only say, that at the North-Eastern Hospital for Children, where we do not admit patients above the age of twelve years, I have had many opportunities of demonstrating this form of murmur.

I have said that you may find difficulties in deter-

* I find, from notes taken by myself, of 57 cases under my own care where I discovered uncomplicated anæmic murmurs, that 15 only were males. The ages were, under sixteen, 7 cases; from sixteen to twenty-four, 18 cases; from twenty-four to thirty-two, 15 cases; from thirty-two to forty, 6 cases; from forty to sixty-six, 4 cases. The arterial anæmic bruit was heard at the base as well as in the carotids and subclavians of both sides in 8 cases; in both carotids in 11 cases; in left carotid and subclavian in 6 cases; in right carotid and subclavian in 4 cases; in right carotid only 1 case; in both subclavians, 2 cases; in left subclavian, 6 cases; at limited area *over* base not propagated up vessels, 16 cases.

mining whether a murmur be inorganic—*i.e.*, hæmic, due to the causes just considered; or organic—*i.e.*, due to structural change of the aortic valves. This difficulty occurs especially when the murmur is localised at the aortic cartilage, and when, as is sometimes the case, a general condition of anæmia develops or reinforces a murmur, due to structural disease. We shall lessen the difficulty when we have contrasted the organic with the inorganic basic murmur.

In the case of the soft murmur, which we are still considering (for we shall hereafter notice the loud basic murmur), the difficulties in diagnosing between the anæmic and the structural may be great. But the cases wherein the chances are in favour of its being structural will be, as I consider, in two classes —one in young subjects, where there has been a history of rheumatism, and especially where there is, or has been, chorea; the other in patients past middle life, where there is a probability of atheroma. In these cases there may be but slight impediment to the onward course of the blood in the aorta. It is very seldom that the aortic valve is much affected by the ordinary form of rheumatic endocarditis without the mitral valve being affected first or coincidentally. Bear in mind that we are now considering a soft murmur, heard alone over the aortic valves, without discoverable alteration of the other valves. There is a condition of the aortic valve in which the edges of its segments are fringed by little villosities, so-called vegetations; these may give rise to a very soft aortic murmur with the first sound. This condition is not necessarily an accompaniment of rheumatism, but where a valve is diseased by rheumatism or by atheroma, it is more apt to occur. In chorea, as we

shall consider hereafter, the vegetations are more frequently found fringing the *mitral* valve, but they do occur on the aortic segments. In patients after middle life, such vegetations may be detached from the valve by the force of the current of blood, and being carried into the arteries, may plug one of the arterial branches. Such may occur in various parts of the system, but especially in the brain. Occurring in the small terminal arteries of certain parts of the brain, it is considered with great probability to be often the cause of chorea; when blocking a larger trunk, it causes various forms of paralysis, or attacks which are ascribed to apoplexy. Remember, therefore, the possibility of arterial embolism when you hear a soft murmur localised at the aortic cartilage in cases at the predisposing ages I have just alluded to, and wherein you do not find anæmia to be a direct explanation of the phenomenon.

So also you may turn the argument round and conclude that when, with these predispositions and under these circumstances you hear a soft first-sound murmur localised over the aortic cartilage, such murmur is probably due to a slight obstruction of the aortic orifice, and that vegetations may fringe the valves.

We will now suppose that instead of the soft murmur we have been considering, there is *a loud bruit heard over a wide area, but having its maximum intensity over the aortic cartilage.* When you commence to auscultate the heart region, you are at once cognisant of a loud rough systolic murmur; over the second right costal cartilage this is very intense; as the stethoscope is carried towards the apex, the sound is found to become less loud, and probably at the apex

itself it ceases to be audible. On auscultating in the reverse direction the bruit is loudly heard in the direction of the aorta and the carotid arteries. In some cases it may be heard at the back, but then only about the level of the spines of the scapulæ. In case of a murmur having these characters, if you have eliminated the probability of an anæmic causation, you will have no difficulty in diagnosing obstruction (stenosis) of the aortic outlet due to a diseased condition of the valves. The concurrent signs will be hypertrophy, without dilatation, of the left ventricle, with small hard arterial pulse. The pathological changes which give rise to this form of murmur consist in thickening, rigidity, and fusion of the segments of the semilunar valves of the aorta, so that the orifice of exit of the blood from the left ventricle is narrowed. This may occur to such extent that the orifice will barely admit a probe. Sometimes the valves are hardened from atheroma and calcareous deposit, sometimes fringed and obstructed by fibrinous vegetations or warty excrescences, sometimes roughened by ulceration.

It is important, even from a diagnostic point of view, to attempt to define under what conditions these various causes of obstruction of the aortic orifice arise. A significant and somewhat strange argument meets us at the very commencement of the investigation. Taking the positive evidence of post-mortem examinations, lesions affecting the aortic valves alone are of very common occurrence. Dr. King Chambers, in analysing 367 cases in which valvular lesions of the heart were discovered at the autopsies, found that the aortic valves were solely affected in 107, the mitral only in 96. The general experience of observers has

been that the frequency of disease in the mitral valve alone, and at the aortic orifice alone respectively is about equal, but that the proneness of the mitral is slightly the greater. Compare this observation with clinical experience; I think you will find all physicians concur in saying that mitral lesions *per se* are met with far more frequently than aortic. Of the hundred cases of heart disease which I mentioned in an early part of these lectures, as observed by myself, only fifteen were declared by the physical signs to be solely due to morbid changes at the aortic orifice, whilst no less than fifty-eight were mitral. It seems fair to conclude from these facts that aortic lesions are often present during life, but are only discovered after death. The regurgitant lesions which we shall soon consider are much more baneful in their *obvious* effects than the obstructive lesions of the aortic valves. It is very probable, therefore, that obstructive lesions are more frequently present than detected. Positive experience confirms this view in many cases, especially of sudden and alarming symptoms in old people with cerebral or visceral disease; patients who are sometimes brought moribund to the hospital are found to present obstruction of the aortic outlet. Now as to the forms of disease which produce aortic obstruction. According to my experience the most common cause in patients who present themselves to us is rheumatic endocarditis. Of twenty-four cases observed by myself in which the physical signs indicated aortic obstruction, ten were obviously rheumatic, nearly all having suffered well-defined rheumatic fever. Flint records that of thirty cases of aortic lesions rheumatism had occurred in sixteen. We shall see, however, that the aortic valves are less

prone to be attacked by rheumatic endocarditis than the mitral. Rheumatic endocarditis attacks first the mitral valve, then extends upwards involving the endocardium lining the ventricle till it includes the aortic valves in the morbid change.

In the majority of cases in which we hear an aortic obstructive murmur without evidence of impairment of the mitral valve, endocarditis has not been limited to the aortic valves, but has begun in the mitral though its effects have not been sufficient to destroy the integrity of the latter. Besides the alteration of the valves from rheumatic endocarditis, there is another cause which is very common in inducing obstruction—atheroma. In this the valves are often incrusted with calcareous deposit. This form of alteration is exclusively met with after middle age, and is by far the most common cause of aortic obstruction in patients past the prime of life. By both these forms of disease (rheumatic endocarditis and atheroma) the narrowing of the aortic orifice may be extreme, and yet the signs of subjective and objective may not be pronounced. In two cases, one mentioned by Stokes and another occurring in America, the orifice left in the fused and hardened valves was so small as only to admit a small probe, and yet disease of the heart was not suspected till the occurrence of acute disease of the lungs which proved fatal. If the signs in such extreme obstructions are obscure, they are still more so in the form of endocarditis characterised only by vegetations upon the valves. According to French observers, these lesions have nothing to do with rheumatism, but are associated with various maladies (Lancereaux, "Anatomie Pathologique," p. 220). It is this form of endocarditis which is especially asso-

ciated with chorea in young subjects, and with the accidents of cerebral embolism in the old. There is yet much obscurity as to its pathogenesis; it is characterised by hyperplasia of the superficial layer of the endocardium, giving rise to little excrescences forming groups upon the ventricular surface of the valves, sometimes fringing their free borders. These vegetations are often attached to the surface of the valve by slender pedicles easily detached; their size is frequently augmented by the attachment of fibrine derived from the blood current. It differs in histological characters from the rheumatic form of endocarditis which affects the deeper fibrous structures of the valves and involves their whole substance. It seems very probable that the French observers are correct in differentiating this form pathogenetically. Lancereaux says that it may occur under various obscure conditions, but especially in alcoholism, the puerperal state, and, perhaps, in intermittent fever. Whether isolable or not, I think we must admit, though M. Lancereaux seems loth to do so, that this form of endocarditis may accompany other forms—in fact that, though it may probably arise *per se* in certain as yet untraced conditions, it frequently occurs upon any valve which has undergone morbid change from other causes.

Another form of disease affecting the aortic valves, which though rare must be borne in mind as affecting diagnosis, is that in which the valves are ulcerated. Under certain circumstances of depressed vitality, the valve already diseased tends to ulcerate; the patient is seized with rigors and symptoms of septic poisoning. The débris of the ulcerated portions of the valves form plugs which, being carried by the cur-

rent of blood, cause embolism of many arteries throughout the system as well as general blood-poisoning. In eleven cases recorded by M. Lancereaux, the aortic valves alone were affected by the ulceration in six. The disease may arise in various adynamic conditions; several cases have been recorded as occurring in the puerperal state. You may conclude that when in a case of aortic obstructive disease (mitral conditions we shall consider hereafter) your patient is seized with severe rigors, with pyæmic symptoms, with vomiting and diarrhœa, with alternations of very high with low temperatures, and especially with signs of cerebral embolism, there is present an ulcerative endocarditis. The disease is uniformly fatal.

To resume concerning aortic obstructions in general, especially as regards prognosis. When you have a loud obstructive murmur with a history of rheumatism or of senile change, with some cardiac hypertrophy, the lesion, compensated as it is by increased force of the heart, need not give occasion to a grave prognosis. The patient may with considerable probability live long in spite of the obstruction. It is in the case of the soft murmur especially that, in my opinion, you should be guarded in your prognosis. Examine your patient with great care, for these cases are often overlooked. Remember in children the proclivity to chorea; in advanced age, in puerperal conditions, in alcoholism and, I think, in Bright's disease, the danger of embolism. When you are called to a case of so-called apoplexy where the symptoms occur *very suddenly*, always carefully auscultate the heart, remembering the great probability that there has been cerebral embolism by detachment of a vegetation from a diseased valve.

We return now to the aortic cartilage. Suppose that we find the first sound to be unaccompanied by murmur but not so the second or diastolic sound. The latter, which should consist only of the click occasioned by the closure of the semilunar valves, is complicated by a murmur. Like the systolic which we have just considered, this may be soft and short, or loud and pronounced. If soft, you will notice, first, its point of greatest intensity. You will probably hear it at the aortic cartilage, but on carrying your stethoscope to the left side of the sternum and auscultating over the cartilage of the third or fourth rib, it will be yet louder. The "reason why" of this we shall presently see. Notice in the next place whether some of the clicking sound of the closure of the aortic semilunar valves is heard with it—*i.e.*, whether it *accompanies and not replaces* the aortic second sound; if this occurs it is evidence that some of the segments of the valve are capable of performing their functions—that the lesion does not involve them all.

This second-sound murmur, instead of being soft and local, may be loud and prolonged; in quality it may be rough, or musical, and it may be heard over a wide area. You may not hear it over the right or left base, or up the great vessels, but by carrying your stethoscope downwards along the centre-line of the sternum you arrive at a spot where the murmur with the second sound is distinctly heard. Exceptionally it is heard only as you get near the apex of the heart. You have many collateral signs to guide you in the diagnosis: the visible throbbing of the arteries, the heaving of the præcordial region, the signs of hypertrophy and dilatation of the left

ventricle. In no condition is the probability greater of enlargement of the heart; in some cases there is enormous increase of mass and weight, the so-called "cor bovinum."

There is another objective sign which I take to be of great value. On making an *ophthalmoscopic examination* of the *retinal vessels* by means of the erect image, you observe *pulsation of the veins or arteries, or of both.* Dr. Stephen Mackenzie has done great service in drawing attention to this sign. I have made ophthalmoscopic examinations in a large number of cases in which an aortic second-sound murmur existed, and I have very rarely failed to find visible pulsation of the retinal vessels. Such pulsation is found most commonly in the veins, but both arteries and veins pulsate in many cases. This sign may have great value, especially when other signs are masked—for example, when the lungs are affected, and the loud rhonchi and râles of bronchitis render it difficult to hear the sounds of the heart.

Suppose that you have heard a second-sound murmur, and that by its position and by the existence of collateral signs you have located its production in the aortic outlet, what is its pathological significance? It means that there is regurgitation of the blood-stream into the left ventricle when the heart is in diastole, on account of the imperfect closure of the valves which guard the aortic outlet. In the case of the systolic murmur, we had *obstruction* afforded to the onward stream of blood through the aortic orifice; in the case of this diastolic murmur we have *insufficiency* of the valves to close the orifice after the systolic gush is over.

Imperfection of the valves permitting regurgitation

into the left ventricle, is brought about by pathological processes similar to those which induce obstruction. In the rheumatic form of endocarditis the valves, after having become thickened by hypertrophy of their connective tissue, undergo a slow and gradual process of contraction, so that the free edges of the valves are retracted from the centre, and a gap of necessity results. Or, in the villous form of endocarditis, a bunch of vegetations may depend from the segments of the valve on their ventricular aspect, and so weigh down such segments as to prevent their apposition in diastole. These masses of vegetation may vary in size from a pin's head to a walnut. Again, such imperfection of the valves as may induce regurgitation, may be caused by the rigidity of segments which have undergone atheromatous change. In rare cases the valves may become ruptured through violence or perforated by ulceration.

In all these pathological conditions, the mechanical effect is reflux of blood, in diastole, through the abnormal gap in the valves. The secondary effects are, first, that there is a deficiency of supply of blood to the general arterial system; secondly, that the left ventricle contains always too much blood. The further consequences are, that the ventricle undergoes compensatory hypertrophy, thus making up for the loss to general arterial system, induced by the reflux, and that, from its over-repletion (for you will understand that it contains blood in diastole when it ought to be empty, and the normal amount of aërated blood is superadded to the amount which has regurgitated through the imperfectly-closed aortic orifice) the ventricle becomes dilated. You will thus

comprehend why, in the case of aortic obstruction, we have hypertrophy only of the left ventricle, whilst in aortic regurgitation we have hypertrophy and dilatation. In obstruction there is enhanced power of the ventricle to overcome the difficulty, but no distension of the ventricle by an abnormal content of blood.

You are now in a position to understand the mechanism of the murmur of aortic regurgitation—it is the murmur caused by the backward rush of blood through the partially-closed and irregular aortic orifice. Remember that this is not a merely passive reflux—it is not simply by the weight of the column of blood that the murmur is occasioned. The ventricular systole drives the blood into arterial channels which are both elastic and muscular. There is a recoil, therefore, of all the arteries which have been distended by the ventricular systole, and the blood is forcibly urged backwards into the ventricle. Thus you may understand how prolonged and loud the murmur is in some cases.

We have now considered three forms of heart-murmur—the aortic direct, or obstructive; the hæmic or anæmic, and the aortic regurgitant or murmur of aortic insufficiency. It will be well to make a short digression and endeavour to understand the physical mode of causation of these abnormal sounds. You will, I think, best arrive at this result by considering that the laws which govern the production of heart-murmurs are precisely similar to those regulating the production of sounds in the case of musical instruments. In wind-instruments the sound is produced chiefly in two modes; in the one by the rushing of air through a contracted orifice, as in the flute and cornet-à-piston, in the other by the vibration of a

tongue of metal, as in the jew's-harp and accordion. The requirement for the production of tone is that a fluid shall be thrown into vibrations of sufficient rapidity, and that these vibrations shall be communicated to the ear. In the case of musical instruments, the vibrating medium is air; in cardiac murmurs the fluid which vibrates is the blood. The blood-stream, being urged through a contracted orifice, is thrown into a number of eddies which are called "fluid veins," and are the source of sonorous vibrations. You know how cooing, whistling, or hissing sounds are produced, by the air issuing from the mouth with varying force, and with varying size of the external aperture, produced by movements of the mouth and teeth.

When a shred of fibrine, or a villous growth, obstructs the stream as it passes through the orifice, this vibrates like the "tongue" of the jew's-harp, and often gives rise to a murmur which has a musical character.

In any condition of the blood when we have contraction of the normal outlet, or where the blood-stream impinges on a vibrating body, a murmur may result. But other circumstances may induce a similar sound. The production of sound may depend on the nature of the fluid medium. It is very obvious that whilst the movement of a viscid fluid gives rise to no appreciable sound, that of a more mobile fluid is loud and pronounced. Compare the effects of treacle and of water respectively poured from a height. When the blood is thin, deficient in red globules, it is more prone to the production of murmurs. Is this condition of anæmia *alone* sufficient to explain the occurrence of the anæmic murmur heard at the base

of the heart and over the arteries? I cannot give a positive answer to this question, but I think *not*. It is true, as Marshall Hall demonstrated, that such murmur may be induced by the abstraction of a quantity of blood from the system, but it is to be recollected that anæmia is not the only condition induced by such depletion. If such murmurs were due solely to the blood condition, they ought to be constant in states of anæmia, and constant in the sites of their production. We often see patients, however, who are very markedly anæmic, and yet who manifest no such murmur; and I have already shown that the sound may be heard at the aortic orifice or over the subclavian or carotid artery of one side, or of both; in fact, in various situations over the arteries. It appears to me, therefore, that we must invoke a concurrent cause to explain the anæmic murmur, and this is present in the condition of the wall of the arterial vessel.

To return to our analogy with musical instruments. A great influence upon the production of musical sound is exerted by the nature of the substance which encloses the vibrating air. In the flute, the material is wood, in the cornet, brass; both media contribute in themselves to the production of vibrations. So in the case of anæmic murmurs, it seems most probable that vibration of the arterial wall may combine with the vibrations readily induced in the watery blood to produce the sound. The pathogeny is not only hæmic but vaso-motor. I shall briefly return to this subject in the consideration of murmurs heard over the right cavities.

A murmur should be heard in the situation superficial to the point where it is generated; this point

in the case of valvular murmurs is the morbidly altered orifice, in the case of anæmic murmurs (on the hypothesis I have just cited), the part of the arterial wall which has lost its tonicity, and has been rendered prone to vibrate. There are, however, two kinds of circumstances which interfere with this direct transmission of the murmur to the ear—which, in fact, alter the site of the audibility of the murmur. Such alteration of site is induced by the *conduction* or by the *convection* of murmurs.

It is obvious that a murmur will be best heard in the direction wherein exists the best conductor of sound. The normal lung, filled as it is with air, is a very bad conductor; when, however, there are condensations of the pulmonary tissue, exudations or solid growths, these may act the part of conductors, and the heart-sounds, normal or abnormal, may be heard in unusual situations. In children, enlarged bronchial glands frequently act as conductors of heart-sounds. In the case of the diastolic aortic murmur, the sternum, which is comparatively a good conductor, causes the sound to be heard in a direction down its centre-line or along its left border. Conduction, then, is the transmission of sound by *still* media, and that in proportion to their facility of transmitting sonorous vibrations.

There is, however, another way by which murmurs are transmitted, that is, by media in *motion*. A murmur is best heard in the direction of the current of blood. This was called by the late Dr. Hyde Salter, the law of "convection of murmurs," and explains away many difficulties. This also may be illustrated by analogy with the air. We know that air is a very bad conductor of sound; on a still day

everything may be silent, but let a breeze spring up, and the sounds, of distant bells for instance, are wafted to the ear The sound is heard only in the direction of the current of air; if the wind change direction it will be no longer audible. In like manner, a cardiac murmur is heard in the direction of the current of blood. In the murmur of aortic obstruction the sound generated at the time of the systole is carried by the blood stream into the aorta and up the great vessels; in exceptional instances the obstructive murmur is heard only in the vessels, and the remembrance of this law of convection will help you to explain such cases. In the murmur of aortic regurgitation, the direction of the current of blood at the time of occurrence of the sound is exactly the reverse of the former. The stream is gushing back into the left ventricle, and the line of convection is downwards from the aortic cartilage to the apex of the heart. In some exceptional cases the regurgitant murmur is only heard near the apex.* These considerations of transmission of sounds, partly by the best conductors, partly by the moving stream of blood, will help you to understand the position and course of audibility of the murmurs which we have discussed.

From the foregoing considerations, I think you will have no difficulty in diagnosing aortic regurgitation. It is not of rare occurrence, though it is less frequently met with than the obstructive lesion: it was present in four of the hundred cases of heart

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* For arguments and illustrative cases, see "Clinical Lectures," by Dr. Hyde Salter, in *The Lancet*, July 24th, Aug. 7th, and Aug. 14th, 1869, pp. 113, 193, and 225.

disease which I have mentioned as under my care. It is most commonly met with at or after the prime of life (of fifty cases noted by Von Bamberger, only fifteen occurred before the age of thirty), and in the male sex, the proportion in recorded cases being three males to one female. The prognosis is generally bad; in the rare cases of youthful patients compensation by hypertrophy of the ventricle may be such that but little trouble is experienced, but when adult life is attained the lesion is one of the gravest that can affect the heart. This is one of the conditions in which *sudden* death may occur, and the subjects should be cautioned against excitement and over-exertion.

Suppose, now, that you hear two sounds at the aortic cartilage, the one with the systole and the other in the diastole. There is a *double murmur*. Or over certain spots whereto, as I have described, an obstructive "bruit" may be carried by conduction or convection, you may hear a first-sound murmur, and over other spots in the direction of the retrograde current you may hear a second-sound murmur. You may conclude in such cases that there is a combination of the two lesions which we have just considered—that there is aortic obstruction as well as aortic regurgitation. This is not uncommon. The semilunar valves, roughened on their ventricular aspect, or rigid and offering an obstructed orifice of exit for the blood, are also imperfectly opposed in diastole. Examples have been recorded in which the first-sound murmur has been followed after a short pause by another murmur, which has been *abruptly terminated* by the second sound. This has been called a *pre-diastolic* murmur; it indicates roughening of the

endocardium of the aorta above the aortic valves, which, in this case, are competent to close their aperture.*

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* *Vide* Flint, "Diseases of the Heart." Second edition, p. 213. Philadelphia, 1870.

LECTURE VII.

AUSCULTATION.

PART III.

Hæmic pulmonary murmur—Pulmonary stenosis—Relation to cyanosis—Tricuspid regurgitation—Ventricular murmurs—Murmurs in chorea—Myocarditis—Arguments concerning cause of non-valvular murmurs—Cardiac phenomena of typhoid fever—Organic mitral murmurs—Causes of mitral lesions—Varieties of lesion—Mitral insufficiency—Mitral stenosis.

We now leave altogether the region of the aortic valves and cross the sternum to the second left interspace and the sternal end of the third cartilage—the area of the valves of the pulmonary artery.

Suppose that you hear a soft murmur with the first sound localised in the pulmonic area. The chances are enormously in favour of this being inorganic—hæmic. Observe all the rules as regards differential diagnosis which I have given you, for the discrimination of anæmic murmurs when heard at the aortic base. Especially notice whether the sound is carried along the left subclavian. If it be strictly localised, the hæmic murmur may be generated in the pulmonary artery. Dr. Flint considers that an inorganic murmur emanates as frequently from the pulmonic orifice as from the aortic. I must say that I have not observed this in my own experience. I have found the soft murmur heard at the left base in by far the

majority of cases, to be distinctly traceable to the left subclavian artery. I would say of a soft systolic murmur localised in the pulmonary area, always assume it to be inorganic or independent of structural change, unless there is strong collateral evidence in favour of its being organic.

Assuming that you have eliminated anæmia as a possible explanation of the murmur, you have yet to consider whether the sound may be produced by the pressure of a tumour upon the trunk of the pulmonary artery. It has been said that tuberculous lung can give rise to such pressure. I cannot admit, however, that the lung-consolidations of tubercle are attended with local increase of bulk constituting tumours capable of *pressure* against the pulmonary artery. I should explain the murmur heard in these cases as due to the conduction by the tubercular consolidations of a hæmic murmur generated in the pulmonary artery. Tumours in the mediastinum, enlarged bronchial glands, cancerous and other growths in the lung may undoubtedly cause pressure against the trunk of the pulmonary artery and give rise to a systolic murmur. All such probabilities of extra-cardial causation you must eliminate by careful examination.

We will imagine that you have eliminated anæmia and pressure on the pulmonary artery as probable causes, and yet you hear a localised murmur over the situation of the pulmonary semilunar valves. This murmur with the first-sound may be soft and heard only at the spot indicated, or it may be loud and heard over a wide area, but distinctly most intense about the third left cartilage. You may be sensible that it is very superficial—generated very close to the

ear. A murmur with these characteristics will, with much probability, be due to *obstruction of the pulmonary artery*, but before completing the diagnosis we will briefly consider the pathological causation and clinical concomitants of such a condition.

In considering aortic disease, I made no mention of congenital defects, because these so rarely affect the aortic orifice that they do not practically influence diagnosis. Aortic defects are not congenital but acquired. It is quite otherwise with pulmonary lesions. Intra-uterine malformations are far more likely to involve the pulmonary artery; moreover, when endocarditis attacks the "fœtus in utero" it is the *right chambers* of the heart that are much more frequently affected. The rule in after life is exactly reversed.

The pathological causes which give rise to obstruction of the pulmonary orifice operate chiefly in fœtal life. Through faulty development the pulmonary artery itself may be contracted in varying degree even to complete obliteration, a blind extremity or rudimentary cord only remaining. Or the semilunar valves may be fused together and may form a membranous, cartilaginiform or cretaceous septum with a circular or slit-like opening. Or the contraction may be below the valves at the apex of the right ventricle—the conus arteriosus dexter—the cause being the shrinking of the muscular tissue, subsequent to inflammation (myocarditis). In one or two cases vegetations like those described in case of aortic disease have been found about the valves. In the rare cases met with as originating at or after adult life atheroma may be a cause or an inflammation due to direct violence to which the right ventricle and pul-

monary artery by their superficial position would seem to be more liable.*

For convenience in clinical diagnosis, I think it best to group the cases according to age.

In infancy the patient will probably be the subject of cyanosis, presenting the characteristics which we have already considered. You will hear the murmur loudly at the base, though in the small area of chest presented in infancy you may not be able precisely to locate it in the pulmonary region. The blueness of surface, however, the venous distension, and the extreme improbability of aortic disease at this period of life, will leave little doubt as to the diagnosis. You may safely infer that a patent foramen ovale or an aperture in the interventricular septum coexists with the pulmonary obstruction, for such is the case almost invariably.

In later childhood you will have more chance of localising the murmur, unless it be exceptionally loud. You must bear in mind, however, that aortic disease may possibly have been developed by rheumatic endocarditis subsequently to birth; therefore the differential diagnosis between aortic and pulmonary obstruction is now necessary. If the bruit be pulmonary, you will probably observe a condition of cyanosis, at least intermittingly, and venous turgescence increased by exertion and by coughing. Moreover, you may be able to ascertain that the right side of the heart is hypertrophied and dilated. There may be venous pulsation. The following case, which occurred under my own observation, affords many points of interest:

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* Von Dusch, "Lehrbuch der Herzkrankheiten." Leipzig, 1868, p. 248.

A little girl (L. S.), aged eight and a half, was admitted under my care at the North-Eastern Hospital for Children, on January 12th, 1873; she seemed weak, and was *excessively pale*, but presented no blueness nor obvious dyspnœa. On auscultating the heart-region a very loud rough first-sound murmur was heard at the base, quite as intense at the aortic as at the pulmonary point. The child had been ailing occasionally ever since birth, but there was no obvious symptom except weakness. She was one of seven children, of those four were living, one had been stillborn, and one died during dentition. The mother was healthy, and could give no account of "maternal impression." I could not help giving a very doubtful diagnosis—it appeared to me that it was quite as probable, from the physical and general signs, that the obstruction was seated at the aortic orifice as well as the pulmonary. As the case progressed the next observed phenomenon was *diarrhœa*, which began on Jan. 13th, and became very persistent and uncontrollable. On Feb. 1st hæmorrhoids were noticed. Progressive enfeeblement occurred, the radial pulse became scarcely perceptible, the hands very cold, while the feet were fairly warm. A week afterwards delirium was manifested; the diarrhœa persisted, and there was much abdominal pain; emaciation continued, the pallor increased, but throughout there was no cyanosis. The child died on March 10th. At the autopsy we found the lowest lobe of the right lung thickly studded with masses of soft *tubercle*, varying in size from a small pea to a large bean. The left lung also was tuberculous at the apex. The large intestine was ulcerated throughout its whole length, and the mesenteric glands were a complete

mass of hard tubercle. We found about an ounce of pale yellow serous fluid in the cavity of the pericardium, and the heart itself very small, pale, and contracted. The aorta and its valves were quite normal, but the pulmonary artery was very small in calibre (diameter four-tenths of an inch), its walls firm and inflexible, so that it resembled the aorta or a systemic artery. It was a pulmonary artery in miniature, with valves minute but perfect. The area of a section across the aorta compared with that of a section across the pulmonary artery was in the proportion of three to one. The wall of the right ventricle was greatly hypertrophied, so that it was thicker at its thickest part than any portion of the left ventricle. The foramen ovale was patent, the aperture being circular, with rounded edges, the communication between the auricles quite unimpeded. There was no imperfection of the septum between the ventricles; the valves were all healthy.

This case shows that we may have a loud murmur of obstruction at the pulmonary orifice without cyanosis. The signs strongly suggested aortic constriction, but there was one chain of circumstances that led up to the diagnosis of pulmonary lesion—the occurrence of general tuberculosis. The persistent uncontrolled diarrhœa and wasting seemed to indicate tubercular ulceration; the post-mortem examination showed tubercle abundantly scattered throughout the body. Now, tubercular changes in valvular diseases of the heart are, for a reason which I will not stay to discuss, for it is yet very obscure, very rare. The great exception to this immunity is in pulmonary obstructive disease. That congenital pulmonary construction predisposes to tubercle has been noticed,

especially by Lebert and Peacock; the observation is valuable as an element of diagnosis.

When adult life is attained, patients who present signs of obstruction at the pulmonary orifice rarely come under our notice. Besides the signs I have already given you, enlargement of the right chambers of the heart may now be more decided, or more readily detected; in addition a pronounced, superficial systolic *thrill* may be felt over the pulmonary area. There may be considerable difficulty in determining whether the pulmonary stenosis be congenital or acquired. Though the congenital malformation is so fatal that only about fifteen per cent. of the subjects reach the age of twenty, some cases attain to a considerable age. Instances are recorded, where the defect was undoubtedly congenital, in which the subjects lived to the age of forty (Kussmaul), fifty-seven (Peacock), and sixty-five (Stölker), respectively. In nearly all cases, however, inquiry will elicit the fact that there has been some respiratory trouble, or some tendency to lividity from infancy or early childhood. On the other hand, in acquired obstruction there may be a history of comparatively recent development of symptoms; a blow may have been received upon the præcordial region which has set up myocarditis, or in a patient past the prime of life you may observe such evidence of degeneration of the systemic arteries as would suggest the probability of atheromatous change in the pulmonary artery.

Murmur with the second sound in the pulmonary area, due to incompetency of the pulmonic valves, may be dismissed in very few words. It is very improbable that you will meet with an instance. I only know of one,

recorded by Hope; in this the pulmonary artery was dilated, and the apposition of the segments of the valve thus prevented.

Double murmur—that is, murmur both with first and second sounds—has been recorded in a few instances, but is very rare. Of course it indicates obstruction, combined with incompetence.

Having finished the exploration of the pulmonary valves, we now complete the investigation of the right side of the heart by auscultating the *tricuspid area*.

You may hear a soft blowing murmur with the first sound limited to the triangular surface occupied by the right ventricle, but most evident at the base of the *ensiform cartilage*. If you have made this observation with care, and have excluded the probabilities of extra-cardial causation, you will conclude that the murmur is due to *regurgitation* through a defective tricuspid valve.

Remember that the conditions in regard to the production of murmur, are now exactly the reverse of those which we have hitherto considered. A murmur with the first sound the site of which is the aorta or pulmonary artery indicates *obstruction* in one of these vessels, the systole urging the blood through the obstructed orifice. A murmur with the first sound at either of the auriculo-ventricular apertures indicates *regurgitation* through such aperture; the contraction of the ventricle whilst impelling, in the usual way, a portion of the content of blood into the vessel of exit, at the same time forces backwards, on account of the gap left by the imperfectly apposed valve, another portion into the auricle.

As regards the right side of the heart, the patho-

logical causes which bring about this result are dilatation of the right ventricle and morbid changes in the valves themselves. Of these the more common is the former. When from any cause there is considerable and continued venous congestion, the right ventricle becomes distended and dilated. Emphysema of the lung may dispose to this condition, but the most common cause of all is disease of the left side of the heart. Dilatation of the right ventricle follows disease of the mitral valve when there is deficient propulsive power in the left ventricle as consequence from cause. The last result of such dilatation of the right ventricle is that the curtains of the tricuspid valve, which under normal conditions were competent to close the auriculo-ventricular aperture during systole, are by the circumferential traction of the walls of the widened ventricle, withdrawn from the centre, so that their edges do not perfectly meet. In systole, therefore, blood regurgitates into the right auricle. The same condition may result from a contracted state of the papillary muscles to which are attached the tendinous cords of any of the curtains of the valve.

The valve may have been altered by rheumatic endocarditis, though this disease very rarely attacks the right side. It is very improbable that you will meet with a case of tricuspid murmur with a history of rheumatic origin without there being concurrent signs of disease of the mitral or aortic valves. There may, however, be a congenital affection of the tricuspid due to endocarditis in fœtal life, when disease of the left chambers is almost unknown.

You must particularly note the concurrent signs of tricuspid regurgitation. These are, first, the evi-

dences of hypertrophy and dilatation of the right side which we have already discussed—the pulsation of the right auricle may be obvious in the second right intercostal space; secondly, turgescence of the veins of the surface and often cyanosis; thirdly, in some cases, venous pulsation evidenced in the jugulars and, rarely, in the liver. Auscultation tells you of the strong and prolonged sound of hypertrophy of the right ventricle and loud accentuated closure of the pulmonary semilunar valves. The symptoms of tricuspid regurgitation are dyspnœa, dropsy, and the distresses of heart-disease in their gravest forms.* It is very important for you to call to your aid these concurrent signs, for whilst they will assist you when you hear a murmur soft and ill-defined as it often is, they will lead you to a diagnosis when there is no murmur at all. *Tricuspid regurgitation without murmur* is far from uncommon; thus differing from mitral regurgitation, the reason being the comparative feebleness of the right ventricle.

If you have arrived at the diagnosis of tricuspid regurgitation, the prognosis is very unfavourable. There is in this condition no chance of compensating change to overcome the difficulty, such as exists in most other morbid conditions of heart. Enhanced power of the right ventricle but serves the more to engorge the venous system by the greater regurgitation; greater strength of left ventricle, if attained, would but increase by *vis à tergo* the tension in the venous system, but it is not attained because the arte-

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* For a very good account of the secondary consequences of tricuspid insufficiency, see Dr. Milner Fothergill, on "The Heart and its Diseases." London: Lewis, 1872, p. 137.

rial supply to the ventricle is diminished by the existing conditions.*

You may possibly hear in the tricuspid area a murmur which does *not* occur with the first sound, but immediately before it. It is a *presystolic* murmur, and indicates *obstruction* of the tricuspid orifice. This lesion, however, in the absence of disease of the other valves of the heart is almost unknown, and its consideration may be conveniently deferred until after the exploration of the mitral area.

Before proceeding to auscultate the mitral area, I shall ask you to consider certain sounds which are heard in the interval between base and apex, and even over the apex itself—sounds which are *not* due to any organic disease of the valves.

I can show you many examples in which the first sound of the heart, heard on auscultating between base and apex—that is, over the right ventricle—is *rough* in its character. The sound is not pronounced enough to be designated a murmur, but the quality of the contraction is not *pure* like that of the healthy ventricle. I have found this to be distinctly the case in some instances of dilatation of the right ventricle, such as one meets with in chronic pulmonary complaints. It occurs also in some cases of anæmia, and is, in my opinion, evidence of a weak right ventricle.

The sound may, however, be more than a mere roughness: it may be termed a soft first-sound murmur. This also may be due to anæmia, especially when the condition of corpuscle-deficiency is associated with dilated right ventricle. That anæmic murmurs are occasionally generated over the right ven-

* Cf. Von Dusch, *loc. cit.* p. 230.

tricle, I have no doubt whatever. In these cases the soft murmur may be heard only as far as the base, or, and this much more commonly, it may disappear at the aortic cartilage, and reappear with reinforcement over the great arteries of the neck, in the manner I have already described; it is lost, however, at the apex of the heart. You may find such murmurs in cases of any profound alteration of the blood, in advanced stages of tuberculosis and in carcinoma.

But, going one step farther in degree, we may find a blowing murmur heard as far as the apex, and even localised at the apex—a murmur which, from its quality and characters, is absolutely indistinguishable from that of valvular disease. The great point, however, which differentiates it from the organic murmur is its temporary or evanescent character; its occurrence corresponds with a definite period in the history of a disease, it fades away and ceases entirely when the disease enters upon a new phase.

Murmurs having these characters have been described as occurring (1) in chorea, (2) in the early stages of certain acute febrile affections.

We will consider the explanations which have been given of their occurrence—first in chorea. Every one will agree that in a certain proportion of patients suffering from chorea there is manifest a cardiac murmur, sometimes at the base, but more commonly at the apex. I have already said that this murmur is in many cases very soft, often difficult to find, and I think it is a fair inference that it is often overlooked. I know I have often overlooked it myself in the early examinations of a patient. Yet, when once it is manifested, it may be observed to be persistent in some cases, whilst in others it may completely disap-

pear, and leave no trace of heart-affection. What is the pathology of this condition? To begin with a matter concerning which there is no doubt—in a considerable proportion of the cases of chorea which have proved fatal, vegetations have been found in greater or less degree fringing the cardiac valves. Organic disease of the valves will undoubtedly, therefore, explain a certain proportion of the cases. Happily, however, chorea is rarely a fatal disease. Will the existence of structural changes in the valves explain the murmur which, unlike that in other valvular affections, entirely disappears? Many observers say no. How, then, do they explain the occurrence of the murmur which is heard over the mitral area? By assuming that there is in these cases a peculiar contraction of the papillary muscles of the ventricle, to which are attached the cords which control the curtains of the valve, or else a peculiar condition of the muscular walls of the ventricle, whereby the apposition of the valvular curtains is prevented, and consequently regurgitation takes place into the auricle. You will understand that this is mere hypothesis, and it seems to me very difficult to accept it. That in chorea the muscle of the heart should in some degree partake of the muscular perturbation which characterises the disease is not impossible to realise, but whilst this would explain irregularities in the time and quality of contraction which undoubtedly do occur, it appears to me incapable of explaining the murmur. If the cardiac muscle behaved in any spasmodic manner, surely the murmur would present strange variations of site and character; sometimes it would be present and sometimes absent, sometimes soft, sometimes loud; whereas it often

presents no considerable variations from hour to hour, day to day, or week to week. Spasm of the papillary muscles I cannot help but reject; and any constant or consentaneous action on their part, whereby they keep open the orifice they are intended to close, seems also to impose a too great demand upon our credulity. Parietal debility of the ventricle seems to me equally difficult to accept: it is quite unproved that any mere debility of the ventricular wall without dilatation of it could induce a condition of imperfect apposition of the valve-curtains. Moreover, there is this objection to all the hypotheses which assume that the murmur in chorea is due to regurgitation through imperfect or irregular muscular action, that in the cases which are not explained by an undoubted organic change in the valves, there are, so far as I have seen, none of the concurrent signs and symptoms of so grave a lesion as mitral regurgitation.

If we exclude the probability of a muscular causation, how shall we explain the non-persistent murmur of chorea? My answer is that I see no difficulty in concluding that such murmurs are always due to some change in or on the valves. The objections taken to this view may be thus stated:—The endocarditis which produces these changes of the valves is a rheumatic endocarditis; only a small proportion of the sufferers from chorea are demonstrably rheumatic; therefore a changed condition of the heart-valves is unlikely. The second objection is the formidable one that endocardial changes of the valves do not pass away, and the murmur which they occasion is permanent. As regards the first of these objections, however, I have already shown how insidious may be the advent and course of the endo-

carditis, even in the form known as rheumatic, and that especially in children, the articular symptoms may be trivial or entirely absent. Moreover, there is no reason to believe that the form of endocarditis occurring in chorea is of necessity rheumatic. It is the endocarditis characterised by the presence of vegetations on the endothelial surface which is met with in chorea; this is prone to occur on valves already altered by rheumatic disease, but it can occur in the absence of rheumatism. It is a mere hyperplasia of the endothelium which may exist in very slight degree, but when it does occur the fibrine of the blood tends to adhere to the thickened spot. The obstruction caused by such a vegetation may readily occasion the murmur; but under the repeated washings of the current of blood, the pedicle itself and the attached fibrine may be washed away suddenly or gradually, and the valve subsequently may present no trace of lesion. I think that there is a very high probability that fragments thus derived may block some of the arterioles supplying the corpora striata or other portions of the cerebro-spinal motor tract, and that thus is produced that form of chorea which is associated with cardiac change. You must not misunderstand me, however, and conclude that in my opinion this is the *exclusive* pathogeny of chorea. It is to me highly probable that just as epilepsy is induced in some instances by actual disease of the brain and in others by reflex irritations, so chorea may be the result, in some cases of direct physical interference with the arterial supply of the motor tract, in others of the peripheral irritations (such as intestinal worms), and probably in others of psychical stimuli such as fright or emotional shock.

I shall teach you, therefore, that when in a case of chorea you hear a murmur distinctly located at apex or base you are to conclude that there is an organic lesion of the mitral or aortic valves.

We turn now to the consideration of the temporary systolic murmur which may be heard over the apex during the course of certain acute febrile affections.

A murmur with the first sound, heard over the apex of the heart but disappearing entirely with convalescence, has been observed in the course of small-pox, erysipelas, and typhoid fever. The phenomenon has been best investigated in the case of the last mentioned disease. In certain of the cases of typhoid, about the end of the second week from the onset, a first-sound murmur may be heard over the right ventricle or just confined to the situation of the apex. When localised at the apex the sound may have precisely the characters of an endocardial murmur, yet as convalescence approaches it entirely disappears. Certain other cardiac phenomena accompany it. The impulse of the heart may be felt to be very feeble or undulatory: the radial pulse may become intermittent; the second sound of heart may be heard to be reduplicate. The signs show great enfeeblement of the heart; this is so obvious that it does not need discussion, and it is well known that some of the subjects die suddenly in syncope.

We come to the inquiry—to what is the first-sound murmur which is heard at the apex in these cases due? We may at once exclude any organic disease of the valves, for it is known to be extremely rare and almost unexampled for endocarditis to develop in the course of typhoid, and the cases which have died in syncope have shown no trace of struc-

tural alteration of the valves. There is evidence, however, of a decided change in the muscular structure of the heart. M. Hayem, who has deeply studied this question, has found evidences of myocarditis—an inflammation of the muscular fibrillæ—with granular and fatty degeneration and a special form known as "vitreous" change. Undoubtedly, therefore, there is structural enfeeblement of the muscular walls of the heart. We have again to encounter the question:—How can such enfeeblement induce the murmur? And first, is there a veritable regurgitation in such cases? M. Hayem considers that there is—the weakened muscle can but imperfectly fulfil its function, the auriculo-ventricular orifice, powerless to resist the force of the blood-current, allows itself to be passively distended, or else the enfeebled papillary muscles can no longer sufficiently restrain the valvular curtains. Hence, a practical, though a functional, insufficiency of the valves.

I must say that I feel considerable difficulty in accepting this explanation. I nevertheless concede at the outset that such difficulty is far less than that which besets the "dynamic" hypothesis in the case of chorea. It seemed to me impossible to admit that a neurosis, the special phenomenon of which is spasm, should give rise to a long-lasting and little-varying condition of patency of the auriculo-ventricular aperture; I even found it difficult to allow that mere passive weakness of the ventricle without dilatation could permit of regurgitation. In the case of typhoid, however, we have to deal not only with general weakness but also with localised lesions. M. Hayem says that in histo-pathological examinations he has found patches of disease in the muscular

fibrillæ disseminated here and there in a most irregular manner.* It is not difficult to assume, therefore, that there is a paralysis of certain of the papillary muscles, and of necessity a condition of incompetence of the valves permitting regurgitation. But then, how shall we explain the speedy restoration? It is very difficult to realise the fact that in a few days such structural lesions shall be annulled. A further difficulty with me is that I find no evidence that the appearance of the cardiac murmur lends gravity to the circumstances. It is surely reasonable to suppose that if in a case of typhoid, with its marked adynamic phenomena, a true and considerable regurgitation of blood at each cardiac systole should occur, the condition of the patient would be of the gravest. It is certain, however, that many patients manifesting such a murmur recover well and promptly, that the respiratory difficulties incidental to the stage of the typhoid are not sensibly aggravated, and that there is no œdema.

Let us consider the lessons derived from a case which has been lately under my own care. A young lady, aged nineteen, whom I saw on the 10th day of well marked typhoid, manifested no cardiac murmur whatever. On the 11th day there was a very soft murmur with the first sound localised at the third left costal cartilage. On the 13th day the bruit reached almost as far as the apex of the heart; it had all the characters of a blowing endocardial murmur; in fact, had I heard it under other circumstances, I am sure I should not have hesitated to ascribe it to mitral regurgitation, for it was heard

* See "Le Progrès Médical," 24 Juillet, 1875, p. 416.

well within the mitral area, though careful auscultation showed that its maximum was a little right of the apex. On the 15th day it was still heard as far as the apex, but its maximum was at the third left costal cartilage. On the 17th day of the fever, the bruit was only heard in the last-mentioned situation, but there was a distinct reduplication of both the first and the second sounds of the heart. On the 21st day there was reduplication of the first sound only, the bruit still audible as before. Convalescence went on most satisfactorily, in a few days all trace of reduplication had ceased, the basic murmur passed away, and slight anæmic murmurs were manifest over the arteries of the neck. On the 38th day the patient called at my house in perfect convalescence, and presenting no signs of cardiac trouble.

This case was one of typhoid of not more than the average severity; the intestinal symptoms were well marked, and the diarrhœa continued during the second week in considerable degree; the pulmonary signs were a slight general bronchitis with condensation about the base of the left lung which soon passed through resolution; the temperature taken in the axilla never exceeded 104·2° F., nor the pulse 128, and defervescence, commencing on the nineteenth day, proceeded with almost perfect regularity. The cardiac troubles seemed to be the most grave of those which surrounded the case, and yet the course of the disease appeared in nowise to be injuriously complicated by them.

Let us review the circumstances of this case with the view of explaining the murmur. In the first place we have positive signs of enfeeblement of the

heart-muscle. A like weakness existed in marked degree in the voluntary muscles; the "subsultus" characteristic of typhoid was a prominent symptom. It is found that in these cases the changes which occur in the muscles of the heart occur also in the muscles of the body. M. Hayem concludes from his able researches that the cardiac fibrillæ are diseased in like manner and in like degree with the fibrillæ of the voluntary muscles. The existence of reduplication of *both* of the heart sounds was a very pronounced sign of cardiac weakness. Let us consider the concurrent conditions. No one could doubt that in a case of this sort there was a profound deterioration of the *quality* of the blood due to the septic influences at work and to the "ensemble" of adynamic conditions. But, furthermore, there must have been a notable diminution of the normal *quantity* of the blood on which the heart could contract, for a part of its volume had drained away (and was still draining) from the alimentary canal, and another part remained stagnant in the congested lung. Such conditions were just tantamount to a notable abstraction of blood.

Cardiac debility and deterioration of blood in quality and quantity, therefore, are *positive* indications in our case. Moreover, we have signs similar to those observed in anæmia. The murmur first noticed could have been taken for an anæmic murmur localised at the commencement of the pulmonary artery. Soon it became heard over the right ventricle; in convalescence it was gradually lost, and anæmic murmurs were heard over the arteries. M. Hayem, speaking from other experiences, says of the murmur, "after having been localised just at the apex

near the nipple, it deviates to the right near the sternum, and tends little by little to ascend towards the base. At the same time the bruit becomes softer, and takes in a more and more decided manner the character of an anæmic murmur."* Now, can we account for the murmur heard at the apex without the hypothesis of regurgitation? Of such regurgitation, remember that we have no evidence whatever except the localisation of the murmur. We have, however, just the factors which induce the anæmic murmurs which are heard over the aorta and the great arteries; these are, as I have before said, a condition of general anæmia and a local enfeeblement of arterial-wall rendering it prone to vibration. Are not these conditions adequate to explain the apex murmur without resort to the regurgitation hypothesis? Instead of passive vibration of arterial-wall, we have here active but tremulous contraction of heart-muscle. In the case quoted, the murmur which I have suggested to have been the product of these two conditions was manifest over varying parts of the right ventricle; at one time it was localised with maximum intensity at the right apex. I can conceive of its being of a similar origin in the left ventricle, or in both ventricles, in those cases in which it is described as having been situated only over the apex.

I am led, therefore, to exclude the "dynamic" theory of regurgitation in these conditions also, and I shall teach you that the first-sound murmur heard over the heart in acute pyrexial diseases is of musculo-hæmic pathogeny.

You will remember, therefore, that it is necessary,

* "Le Progrès Médical," 24 Juillet, 1875, p. 415.

whenever in a case of pronounced anæmia you hear a soft, first-sound murmur localised at or near the apex, to consider the probability of its occurring quite independently of any implication of the valvular apparatus. And especially if you hear such a murmur in a patient manifesting typhoid fever, if there be evidence that no mitral lesion has taken place previously to the occurrence of the fever, you may predict with the highest probability that the sound will disappear, and that the valves will not be affected.

These conditions excluded, we now suppose that *a murmur is heard in the mitral area.* You have determined the situation of the apex-beat, and you observe that there is a bruit localised within, or having a maximum intensity within, the circumference of a circle extending an inch around the apex. The phenomenon indicates a morbid condition of the mitral orifice.

A murmur indicative of mitral lesion is met with as probably the most common of all the signs of heart-disease. Of the hundred cases of the various clinical forms of disease which I have cited, fifty-eight manifested the murmur indicating morbid change at the mitral orifice.

By far the most common cause of mitral lesions is rheumatic endocarditis. Of the fifty-eight cases just mentioned thirty-two had suffered from rheumatism, and of these twenty-three had been the subjects of rheumatic fever; two, in addition, had suffered from rheumatoid symptoms which occur subsequently to scarlatina. In seventy cases of mitral lesions noted by Dr. Flint, rheumatism had occurred in fifty-five. You will remember that I called your attention to the frequency of pericarditis in acute rheumatism; a like

frequency exists as regards endocarditis—in fact, the latter probably occurs rather more frequently than the former. Whilst Hasse, Bamberger, and Lebert give twenty-two, twenty, and seventeen per cent. respectively as the proportion of cases of endocarditis occurring in the course of rheumatic fever, Fuller and others have fixed the percentage much higher. Not at all infrequently the two diseases, endocarditis and pericarditis, occur together during acute rheumatism. I have already hinted that endocarditis, like pericarditis, can arise and run its course in subacute rheumatism, where the articular symptoms are very slight indeed—in fact, that in some cases objective symptoms may be entirely absent. We must conclude, therefore, that endocarditis of the rheumatic form may occasionally occur very insidiously, so that its origin and course may be entirely overlooked.

Rheumatic endocarditis commonly starts from the mitral valve; in many cases the disease does not extend further. It is an obvious corollary that the mitral is the most frequent site of valvular deterioration. Combining the figures of Willigk, Flint, and Cockle, it would appear that the mitral is affected in one hundred and sixty-six cases to one hundred and thirty in which the aortic valves are diseased. The disease spreads from the valve to the endocardium lining auricle and ventricle; its effects may often be traced by the appearance of a milky patch of thickened endocardium, stretching in a direct path across the ventricle from the mitral to the aortic valves. The pathological changes occurring in rheumatic endocarditis consist, first, in a swelling and thickening of the serous membrane and the substance of the valve, the microscope showing an increase in the number of con-

nective tissue nuclei; subsequently there is much development of fibrous tissue; lastly, there is a gradual process of retraction of the newly-formed tissue just as occurs in cicatrices. The morbid process may involve the muscular structures immediately subjacent—there may be myocarditis, with the result of shortening of the papillary muscles and consequent retraction of the cords and curtains of the valve.

Other causes besides rheumatic endocarditis may produce lesion of the mitral valve and orifice. These are identical with those which we have already considered as affecting the aortic valves—atheroma and endocarditis of the villous (*i.e.*, accompanied by vegetations) and the ulcerative forms. The valve may be incrusted by calcareous salts, and rendered hard as bone. Endocarditis may be induced by renal disease. Lancereaux has recorded a very interesting case, in which, with a condition of contracted kidney, there was disease of the mitral valve characterised by thickening and the appearance of vegetations which, under the microscope, were seen to be studded with granules, distinctly proved to be deposits of *urates*. The probabilities are very great that rheumatic endocarditis is due to the presence or excess in the blood of the acid products of tissue disintegration, and that the endocarditis met with in renal disease is due to retention of the products which the kidneys are unable to excrete. In the villous form of endocarditis little excrescences may be seen fringing the margin of the mitral orifice, especially on the auricular side; these have been often observed after death in cases of chorea. The indications of ulcerative endocarditis we have briefly considered in connexion with the aortic valves; remember that it may

in like manner attack the mitral; recent observations have shown that it may occur as one of the sequelæ of parturition. Another cause of lesion of the mitral orifice is rupture of the tendinous cords connecting the curtains of the valve with the papillary muscles. This is not very uncommon; Flint found it in four out thirty-nine cases of mitral defect; it may occur from violent action of the heart. Again, the mitral orifice may be rendered imperfect on account of dilatation of the ventricle, whereby approximation of the curtains of the valves is prevented. Lastly, but very rarely, fusion together, or perforations of, the mitral valve may be congenital.

When we come to review these various pathological causes in relation to their effect upon the auriculo-ventricular orifice, we find that they can produce two well-marked varieties of lesion as well as a third variety which is a compound of both the others. Thus the orifice may be *patent*, the valve imperfectly closing it; or it may be *obstructed*, the outlet from the auricle being narrowed; or it may be both *obstructed and patent*, an impediment existing to the outflow from the auricle, as well as an imperfection of the valve whereby it is prevented from closing the orifice.

The first of these conditions is the most common. By thickening and shrinking of the curtains of the valve, by the presence of vegetations distorting them or weighing them down, by their "pouching" with aneurismal dilatations, by their perforation or ulceration, by swelling and shortening of the tendinous cords attached to the curtains, by deformity or rupture of the musculi papillares, by disease and retraction of the muscular wall of the ventricle, or by such

dilatation of it that the edges of the valves cannot meet—by these and some other causes the mitral orifice is prevented from closure at the time of the systole of the heart. There is then said to be a condition of *mitral insufficiency;* that is, the valve is insufficient to close the auriculo-ventricular aperture. The consequence is that at each contraction of the ventricle a portion of the content of blood gushes backwards, through the space left by the imperfect apposition of the valve, into the left auricle. There is said to be *mitral regurgitation.* One consequence of this condition is that the auricle is always abnormally full; the continuance of this leads to *dilatation of the auricle,* and the amount of such dilatation affords an index of the amount of regurgitation. Furthermore, the ventricle is of necessity incompletely emptied; from containing habitually too much blood it also becomes *dilated,* and, in obedience to the law which enables involuntary muscle to increase in bulk and strength in order to overcome obstacles, it becomes *hypertrophied.* This condition of hypertrophy with dilatation has been called eccentric hypertrophy. The consequences on the general system arising from the leakage through the mitral orifice are, unless the condition of hypertrophy exactly compensate for the evils, insufficient supply of blood through the aorta, whence diminished blood-pressure in the arteries, sluggishness of flow in the capillaries from impaired *vis à tergo,* and undue repletion of the venous radicles and the general venous system. And when we come to the right heart we find a condition of distension (the pulmonary second sound is intensified, owing to the heightened blood-pressure, as I have before pointed out) and the right cavities may become

dilated. In the majority of cases the cause inducing the condition of mitral regurgitation is *rheumatic endocarditis.* Mitral regurgitation occurred in forty-eight out of my hundred cases of heart disease. Of these twenty-six had suffered rheumatism, nineteen having had rheumatic fever; besides, two had suffered scarlatina, wherein you know there are rheumatoid phenomena. Chorea existed in two cases, gout occurred in one. In three it seemed probable that the condition was congenital.

A quite different set of conditions obtains when the mitral orifice is obstructed. The average circumference of the normal orifice, according to Bizot, is four inches; its form is oval, the long diameter being one inch. These dimensions are greatly modified by disease. The cords, curtains, and muscles of the valve may be stiffened into a rigid mass, or the edges of the orifice may be obstructed by vegetations, in some cases very small and insignificant, in others assuming the dimensions of large polypi. In many cases the form of the valve-curtains and the shape of the outlet are singularly altered. The curtains are, as it were, fused together into an even tube, more or less conical, with its smaller extremity downwards opening into the ventricle: sometimes this extremity is circular and may be extremely small; it may admit only the thumb or the little finger, or be so minute as scarcely to allow a small catheter or even a crow's quill to pass through it. This variety is known as the "funnel-mitral." Or the free extremity, instead of being circular, may be slit-like. Dr. Hilton Fagge has recorded a case in which the slit was so narrow that it would not admit a threepenny-piece edgewise. This is called the "button-hole" mitral. The septum formed by

the adherent valve-curtains may be incrusted with calcareous salts so as to be of bony hardness. In many cases, however, it is perfectly smooth and so uniform in its conformation as a hollow cone, that it seems to suggest that it must be a congenital anomaly; its perfect regularity appearing to contradict the probability that it is the product of disease.

The effect of these conditions upon the auriculo-ventricular aperture is the reverse of that produced by the conditions which permit regurgitation. The aperture guarded by the mitral valve instead of being widened is narrowed—there is said to be *mitral stenosis*. There exists an impediment to the flow from the auricle into the ventricle—there is *mitral obstruction*. Such an occurrence is not uncommon. Flint found stenosis in sixteen cases out of thirty-nine instances of mitral lesion. Of my forty-eight cases of mitral disease, stenosis was declared by the physical signs to exist in ten.

As I have before said, the regularity of form in the case of the fused mitral valve has suggested the probability of a congenital causation. Dr. Hilton Fagge inclines to this view in some cases, and thinks that a rheumatic origin is comparatively rare. For my own part, however, I am disposed to the view that nearly all the cases are the result of rheumatic endocarditis of a very chronic form. Of the ten cases I have mentioned, four had suffered from rheumatic fever, and two from subacute rheumatism; in the remaining four the causation was hypothetical. In undoubtedly rheumatic cases, however, I have seen the funnel mitral of the precisely regular conformation suggestive of the congenital anomaly, and I think it must be explained by the involvement of the

whole texture of the valve by one or successive attacks of endocarditis (which attacks, as I have before said, may occur with little or no subjective sign), and by slow and regular development of fibrous tissue with quasi-cicatricial change. The great rarity of the positive evidence of congenital anomaly of the mitral valve is in favour of this view.

The secondary effects of mitral obstruction are quite different from those of mitral regurgitation. The most constant and most pronounced effect is upon the left auricle, the wall of which becomes *hypertrophied.* I have already cited a case under my own care illustrative of this point in considering the chronometry of pulsations. It is quite clear that the hypertrophy occurs in obedience to the usual rule—there is an impediment to the outflow from the auricle to the ventricle, and the muscle of the auricle becomes hypertrophied to overcome the obstruction. Whilst dilatation of the left auricle without hypertrophy is characteristic of mitral regurgitation, dilatation *with* hypertrophy is characteristic of mitral obstruction. The left ventricle in mitral obstruction is found not to be dilated. In some cases it has been observed to be smaller than normal, its muscular wall has rarely been found hypertrophied. When it has been thus found the condition has been that formerly known as *concentric* hypertrophy, which simply means hypertrophy without dilatation. In the great majority of cases the left ventricle in mitral obstruction is found not obviously abnormal, the condition thus differing from that in mitral regurgitation wherein dilatation and hypertrophy are the rule.

The consecutive changes in the right heart are alike in regurgitation and obstruction, though the

initial causes are different. In both conditions there is impaired *vis à tergo*, and hence venous congestion and dilatation of the cavities of the right heart, but this is due in the case of stenosis to the imperfection of supply from the auricle to the ventricle with retention in the auricle, whilst in regurgitation it is due to the retrograde diversion of the blood-stream through the mitral aperture.

The subjoined diagrammatic sketches may aid you to comprehend the conditions in mitral stenosis, as distinguished from those in mitral regurgitation. They are intended to represent vertical sections through the left auricle and ventricle.

FIG. 3.

MITRAL STENOSIS (Obstruction).

a, Hypertrophied muscular wall of left auricle.
b, Section of wall of left ventricle.
c, Narrowed auriculo-ventricular orifice.

FIG. 4.

MITRAL INSUFFICIENCY (Regurgitation).

a, Dilated left auricle.
b, Section of hypertrophied and dilated left ventricle.
c, Patent auriculo-ventricular orifice.

(The arrow shows the direction of the current of blood at the time of the production of the murmur.)

LECTURE VIII.

AUSCULTATION.

PART IV.

Differential diagnosis of mitral lesions—Rhythm—Chronometry of murmurs—Vocal representation of murmurs—Graphic representation of murmurs—Area of audibility—Signs of mitral stenosis—Signs of mitral insufficiency—Double mitral murmur—Combined murmurs—Diagnosis of complex lesions.

SEEING that there are these well-marked varieties of lesion of the mitral orifice, the question now occurs—can they be detected and differentiated during life? The answer is undoubtedly in the affirmative.

The previous examination of the patient has given many data. We must exclude the evidence of any departure from the normal as regards the right chambers of the heart, for consecutive changes in these can occur from either of the abnormal conditions of the mitral orifice. Our examination of the left chambers, however, may have afforded valuable evidence. If we have found from the physical signs that the left ventricle is dilated or dilated and hypertrophied, the probability is great that if there be any mitral lesion at all it will be one of *regurgitation.* Palpation may strengthen this probability by evidencing a systolic thrill; remember, however, to time it carefully, for systolic thrill is rare and presystolic thrill by far the more common. Suppos-

ing, on the other hand, that we find little or no evidence of displacement of the apex, but a thrill communicated to the finger distinctly antecedently to the impulse of the heart—in this case the diagnosis of mitral *obstruction* may be positively made. In some such cases, as I have before shown, the pulsation of the hypertrophied left auricle may be visible in vibration of the chest-wall and its movement immediately before the ventricular systole can be demonstrated.

It is but a small proportion only, however, of the cases of obstruction of the mitral orifice that can be thus diagnosed. The question now becomes narrower: can the conditions be differentiated by auscultation? Fifteen years ago it would have been declared impossible. Contraction of the mitral orifice was revealed by post-mortem examinations in a large number of cases then as now. The diseased condition of the valve was not overlooked, but obstructive and regurgitant lesions were no doubt mingled together in clinical diagnosis. A murmur was heard in both classes of cases, but the rhythm of such murmur was not recognised. Although some steps had been taken in this direction by French observers, Fauvel and others, it was not till Dr. W. T. Gairdner, in 1861, by careful clinical observation and philosophical demonstration showed the characters, the import and the causation of the murmur dependent upon mitral constriction that the diagnosis in any considerable number of cases was effected. Further observations, especially those of the late Dr. Hyde Salter and Dr. Hilton Fagge, have contributed to spread the knowledge of the methods of discriminating the two conditions, but there is not

the least doubt that even now the condition of mitral stenosis in a large number of cases is not differentiated from the regurgitant lesion. As to the ease with which this diagnosis can be effected the opinions of good observers vary; some say, with Dr. Hyde Salter, that any one who should fail to recognise the murmur of mitral obstruction "could hardly be considered a decently informed member of our profession;" others, with abundant opportunities of observation, have failed to discover the murmur, and have written papers to prove that it has no existence. The truth lies probably between these two extremes. The typical presystolic murmur any instructed clinical observer who takes the necessary time and trouble cannot fall to recognise, but there are some murmurs so short, so slight, and so obscure that their recognition and the determination of their rhythm are matters of very great difficulty. Such obscurities are, however, quite exceptional, and in the great majority of cases it will be your own fault if you fail to diagnose mitral stenosis when it exists.

We must first inquire: what is the rhythm of the murmurs heard at the apex of the heart? At the base we know that the rhythm is very simple. Of the murmurs there localised one is heard with the first sound (systolic), the other commences with the second sound (diastolic). At the apex we may have, as at the base, a murmur coincident with the first sound—systolic murmurs may occur both at the base and apex—but, as a matter of fact, a murmur whose origin is at the mitral orifice never commences at the same period of the heart's rhythm as the diastolic murmur heard at the base. A mitral murmur never occurs *with* the second sound, but *after* it. There is

no such thing as a diastolic murmur heard at the apex. In times past such a murmur was described as an exceptional occurrence, but careful observation will show that a mitral murmur cannot be diastolic in the sense of being absolutely coincident with the diastolic or second sound of the heart, but that it occurs *after* the second sound and *before* the first sound. It is now well known as the presystolic murmur. I have already described to you the mechanism of presystolic thrill and presystolic pulsation. The murmur which we are now considering is produced by the same cause—the muscular contraction of the left auricle forcing the blood through the narrowed auriculo-ventricular orifice.

The term *presystolic* is not wholly devoid of objection. It is obvious that the systole of the heart means the systole both of auricles and ventricles—the presystolic murmur is presystolic only as regards the ventricular systole, it is coincident with the auricular systole. Nevertheless, the term has been so useful that we cannot wholly discard it. Dr. Gairdner proposed the term *auricular-systolic*, and this is perfectly expressive of physiological causation. The propriety of the terms will, I consider, necessarily vary as these are employed for the expression of the *clinical* or the *pathological* conditions. For the latter purpose, Dr. Gairdner's term is perfectly appropriate; but for clinical record it is better, in my opinion, to use a term which shall fix the period of the murmur in the heart's rhythm without even the semblance of hypothesis. I do not think that the use of the words "systolic" and "diastolic," for the purpose of timing murmurs, is without reproach; but these, as well as "presystolic," have become incorporate with our

notions, and we cannot well do without them. We use them, however, on the principle expressed by the phrase: If you know what I mean, what does it matter what I say? For precision, it would be much better, in my humble opinion, if heart-murmurs were expressed in plain English and indicated by the periods of the obvious and precise sounds of the normal heart. Thus, a systolic should be expressed as a *first-sound* murmur, a diastolic as a *second-sound* murmur, a presystolic as a *before-first-sound* murmur.

From this preface let us turn to the practical methods of discriminating the rhythm of the murmurs heard over the mitral area and dependent upon disease of the mitral valve. Here let me say, that you must give full and long attention, for the diagnosis is easy if you take sufficient care, confused and obscure otherwise.

First. Endeavour to determine the exact position of the murmur in the heart's rhythm. We may call this the method of CHRONOMETRY OF MURMURS. The principles of it I have already applied in the cases of pulsations occurring over the cardiac area and the phenomena of thrill. At the risk of frequent repetition I will again state the rule:—Apply your stethoscope over the point of maximum loudness of the murmur, and place the tips of the fingers on any point of the chest where you can feel the impulse of the ventricles or over the site of the pulse of the carotids. You auscultate at the same time that you feel the pulsation of the heart or of one of the great arteries near it. The question which you have to ask yourself is this: Is the murmur which I hear coincident with the first sound of the heart, or is it previous to it and directly terminated by it? Do not

run away with the notion that without all this care you can determine the question; you may not be able even to distinguish the first sound from the second sound. What says one of the greatest authorities living, Dr. Stokes?—"So great is the difficulty, that we cannot resist altering our opinions from day to day as to which is the first and which is the second sound."* There can, I think, be no doubt that the great reasons why in times past the condition of mitral stenosis remained undistinguished from that of regurgitation, was that the murmur was judged to be systolic, and the first sound was taken for the second sound. In cases of stenosis, the mistake is peculiarly easy, for the first sound is usually very short, sharp, and sudden, much resembling the second sound. These considerations are quite enough to inculcate lessons of care in observation, and the absolute necessity of timing the murmurs. If the murmur be systolic you will hear it commence at the same instant that the finger is sensible of the cardiac pulsation. It is produced, as you know, by the reflux current forced into the left auricle by the ventricular contraction. It is a truism, therefore, to say that its period of production is the period of its cause—*i.e.*, the ventricular systole. Commencing immediately with the full force of the contraction, it may last throughout the whole systole, or it may become feebler and inaudible as the ventricle becomes emptied. In other words, it may be short or long; commencing with the systole, its duration may be the whole or a part thereof. Such is the

* "A Practical Treatise on Diseases of the Heart." 3rd edition, 1862, quoted by Dr. Fagge, *loc. cit.*

first-sound murmur,—the murmur of mitral regurgitation.

Suppose now that the murmur which you hear be not coincident with the systole. At the moment that your finger becomes sensible of the impulse of the heart against the wall of the chest, the flap or thud of the first sound is audible. Preceding this, however, and soon after the second sound, a rough murmur is heard. This murmur also may be either short or long; it may commence nearly immediately after the second sound, or it may occur momentarily before the first sound, but the latter always terminates it as with a sudden full stop. The mechanism I have before explained—it is the sound produced by the effort of a hypertrophied auricle in urging the blood through a narrowed mitral orifice. This presystolic, before-first-sound murmur, is absolutely diagnostic of mitral stenosis.

Secondly.—In order to help you to realise the distinctive characters of these sounds, I would call your attention to a method of VOCAL REPRESENTATION OF MURMURS. The "bruits" heard over the heart-region have received since the time of Laennec many names according to their nature and quality. Thus we have bellows sounds, filing, grating, rasping, croaking, crowing, whining, caterwauling, and blubbering sounds, or musical or sibilant sounds. Bouillaud imitated the character and pitch of the sounds by letters, the pronunciation of S expressing the extreme of sibilant murmurs of high pitch, R representing those of low pitch. These illustrations are all of some value, as enabling one to note the variations in character of any given murmur from time to time, and thus indicating changes favourable

or unfavourable, or the persistency of the lesion. Any of the illustrations just given may be applied to the murmurs produced by the valvular conditions we have as yet considered; the sounds, therefore, cannot be considered diagnostic. Valuable help, however, may be derived from a consideration of the sound in the differential diagnosis of murmurs heard over the mitral area. The murmur of regurgitation may have almost any of the characters just described, but not so the murmur of obstruction. This latter is almost invariably rough; it has been called a "churning," "grinding," or "blubbering" murmur. In my own opinion, in typical cases, it may be best illustrated as "rolling" or "bubbling," resembling the sound of air rising through water. You may easily realise its distinctiveness from the systolic murmur by these simple considerations—the systolic fades off but never terminates abruptly, the presystolic always stops suddenly. I have frequently illustrated this by the subjoined vocal illustration, the sound of "*p*" indicating the abrupt termination of the presystolic murmur.

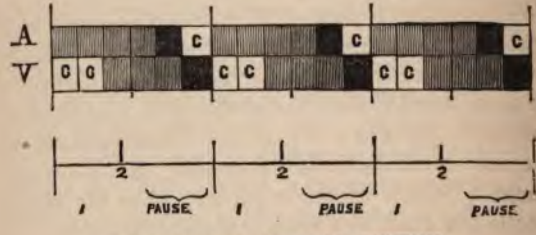
Sound of Systolic Murmur.	Sound of Presystolic Murmur.
Hooh—hoof—ruff.	*Up—rup—rr. rr. rup.*

Thirdly.—We come to a plan which we owe to Dr. Gairdner, and I know of none which is more valuable to fix upon the mind the phenomena of the murmur, and to serve as a method of record. We may term this the method of Graphic Representation of Murmurs. To represent all the facts and conditions, I have combined Dr. Gairdner's plan with a chart which I have modified from Dr. Salter.

The upper portion of this diagram represents the

conditions of the auricle and ventricle during three cardiac pulsations. Each such pulsation or cycle is divided into six equal parts, which are denoted by the squares. The squares which are shaded denote that the chamber (auricle or ventricle) contains blood, whilst the black squares indicate that it is replete—*i.e.*, at its maximum of distension. The squares marked C denote contraction or systole. The upper line of squares A pertain to the auricle, the lower V to the ventricle. Taking the auricle first and proceeding from left to right, we see that it is receiving

FIG. 5.



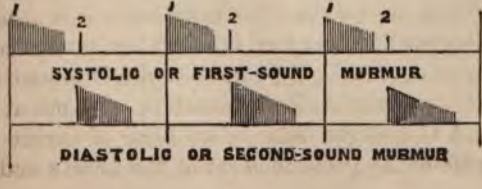
THE NORMAL CARDIAC RHYTHM.

blood during the period indicated by the first four squares, and that at the fifth it has arrived at its maximum distension. Then follows C, its systole or emptying through muscular contraction. Looking at the ventricular line V, we see that the systole lasts during the first two squares, or two-sixths of the cycle, that during the following three it is in diastole or receiving blood, and at the sixth it has arrived at its maximum distension. So the cycle is in each case repeated. Now, we are enabled by a glance at the diagram to note the relative condition of auricle and ventricle at any given moment of the heart's action;

we have only to compare the upper sections with the lower. Thus we see that whilst the ventricle V is in systole, the auricle A is in diastole: that A, having at last become distended, contracts, while V is yet in diastole, and completes the repletion of V, which recommences the cycle by contraction. Now, by a horizontal line below, we have a means of comparing these conditions with the sounds of the normal heart, the vertical lines 1 and 2 denoting the first and the second sounds respectively. You will see that the latter occurs very soon (about half a square) after the ventricular contraction, the first sound lasting during two squares.

We have thus a very simple and concise method of registering murmurs, their position in the cardiac rhythm and their duration. Taking the horizontal line to express the duration of the cycle, and the vertical lines to denote the first and second sounds, we have merely to indicate by shading the place and estimated length of the murmur. Thus the simple systolic and diastolic murmurs heard at the *base of the heart*, would be indicated by the subjoined diagram (fig. 6). The one commences with the first, the other with the second sound.

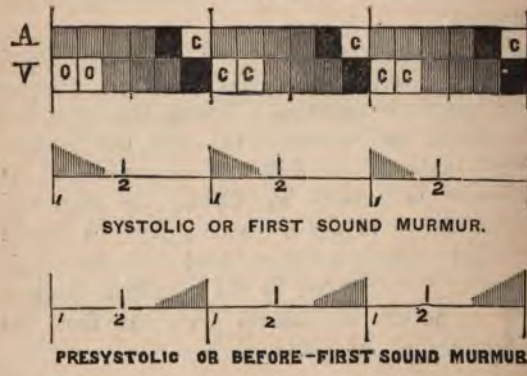
Fig. 6.



The rhythm of the first-sound murmur heard at base and apex is precisely similar. The upper line of this diagram would alike express the sounds of aortic obstruction and mitral regurgitation. Of course a note of the area of audibility and maximum intensity would at once indicate the diagnosis.

We will now consider how this method illustrates the differential diagnosis between the murmurs of mitral regurgitation and mitral stenosis. The subjoined diagram you will readily understand from the

Fig. 7.



explanation I have already given. It shows how the *systolic* murmur starts *from* the first sound, and the *presystolic* leads up *to* the first sound; whilst a glance at the upper section of the diagram indicates the cardiac conditions at the time of the production of the murmurs. The first-sound murmur occurs with V C C, the ventricular contraction; the presystolic with A C, the auricular contraction. It is possible that, as the diagram shows, the presystolic murmur

may commence during the distension of the auricle, and previously to its actual systole; though I am inclined to think that by far the most probable cause of a *prolonged* presystolic murmur is the hypertrophy of the auricle, the systole of which occurs earlier than under normal conditions.

Having considered the *rhythm* of the murmur, we have yet another aid towards the differentiation of mitral stenosis from mitral regurgitation in the determination of the *area of audibility*.

It is rare for the murmur of stenosis to be heard to any considerable degree below or outside the situation of the normal apex. As I have before said, the conditions of hypertrophy and dilatation of the left ventricle, which causes the apex-beat to be discernible externally and inferiorly to the normal position, seldom coexist with mitral stenosis. A murmur, therefore, heard left of the cardiac area in conjunction with signs of ventricular enlargement is unlikely to be a murmur of mitral obstruction. Exceptions to this rule occur when hypertrophy of the ventricle from other causes coexists with mitral stenosis. I have recorded a case in point in which there had been repeated attacks of pericarditis with numerous adhesions.* Observers have agreed that the murmur of obstruction is a localised murmur—it is seldom audible far from the apex. Again, it is said to have its maximum intensity *at* the apex. To this last conclusion, however, I shall have to demur; it has been an almost invariable experience with me that the presystolic murmur is loudest at a point internal to (*i.e.*, right of) the apex-beat. It is usually

* Vide *Medical Times and Gazette*, June 10th, 1874, p. 34.

audible over a portion of the normal area of the right ventricle, but seldom as far on the pulmonary cartilage, but is suddenly cut off at the left apex and is inaudible, or scarcely audible, left of the apex.

Let us contrast this with the murmur of mitral regurgitation. This systolic murmur may have a very small area of audibility around the apex, but its maximum is *never* internal to this point. On the contrary, in a large majority of cases it is louder a short distance outside the apex than at the apex itself. When it is of considerable intensity, the sound of the murmur may be carried towards the axilla, and may be audible over the axillary part of the chest-wall: it will probably disappear as the stethoscope is carried to the back, but may again become evident between the scapulæ, especially in the interval between the angle and the spine of the left scapula.

A little consideration will, I think, enable you to realise the physiological causes of the differences in site of the two murmurs. A reference to the diagram illustrating the conditions in *mitral stenosis* (p. 155) will remind you that the direction of the current—*i.e.*, the line of convection of the murmur, is from the auricle *to* the apex of the heart. It would seem, therefore, *primâ facie* that the "bruit" should be most audible *at* the apex. We must recollect, however, that at the time of production of the murmur the apex is not close to the wall of the chest; its position is slightly internal to that which it occupies when the ventricle strikes the chest-wall, and a space intervenes. These considerations to my mind explain the position and the limitation of the murmur. In *regurgitation*, on the other hand (see fig. 2), the direc-

tion of convection is contrariwise; you might say that the sound ought to be carried to the region of the left auricle. Why is it not so? Surely because the walls of the left ventricle conduct the sound in a direct line to the apex, and thence to the ear. At the moment of production of the first sound the apex is in direct contact with the chest-wall, and occupies a position left of that which it occupied previously. The muscular ventricle, the thoracic parietes and the stethoscope, being in close apposition, constitute one solid conductor through which the sonorous vibrations travel in uninterrupted course to the ear.*

We will now sum up our auscultatory evidence.

A rough murmur is heard antecedently to the first sound, and abruptly terminated by it; its maximum intensity being slightly internal to the normal apex. The condition is one of MITRAL STENOSIS.

We will devote a short time to the clinical history of these cases, and the phenomena which you may observe during your observation of their progress. The late Dr. Hyde Salter noticed the remarkable proclivity of the subjects of mitral stenosis to *hæmoptysis*. He recorded this as occurring in six out of eight cases. I have no doubt, however, that this was an exceptional frequency; for out of sixteen cases recorded by Dr. Fagge, I find hæmoptysis noted only in three, and in seventeen cases under my

---

* Both these murmurs may be intensified by causing the patient to use muscular exertion; but especially the presystolic. The latter is apt to vary in intensity and audibility: it may be absolutely inaudible till the patient is put through a little walking exercise.

own care it occurred also in three. Though these figures modify those of Dr. Salter, they support his conclusion that hæmoptysis is a frequent symptom in mitral obstruction. The cause is no doubt the backward pressure exerted by the contraction of the auricle (opposed as it is by the obstruction at the auriculo-ventricular outlet) upon the pulmonary veins; hence there is congestion of the pulmonary capillaries to the point of rupture. In mitral obstruction the capillaries suffer the *direct* pressure of the contracting auricle, whilst in regurgitation the auricle intervenes as a dilatable cavity between the contracting ventricle and the pulmonary veins.

The next point of interest in the clinical history of these cases is the proneness to embolism of one of the cerebral arteries, or of some artery of the lower limb. Remember, therefore, to inquire carefully into the cerebral conditions of all patients whom you find to manifest the signs of mitral stenosis, and conversely in cases of *sudden* paralysis, apparently of cerebral origin, be mindful to explore the mitral region. You will readily understand how the contraction of the auricle may detach a pellet of fibrine or a pediculated vegetation from the endocardial surface, or from the margin of the auriculo-ventricular orifice, and transmit it into the direct current of blood urged by the ventricle into the arteries.

With regard to other symptoms, the subjects of stenosis are in most respects affected in like manner with the subjects of insufficiency. As I have said before, the secondary effects upon the right chambers of the heart are alike in both cases. So we have in both cough, dyspnœa, and the evidences of con-

gestion, pulmonary and general, and in the end, dropsy, &c. In one point, in my experience, the cases are slightly different—the subjects of stenosis are more liable to variable symptoms and spasmodic troubles.

Mitral stenosis excluded, we turn to the other condition.

A murmur is heard with the first sound, or entirely occupying the place of the first sound in the cardiac rhythm; its maximum at, or external to, the position of the apex-beat. The condition is that of MITRAL INSUFFICIENCY.

In these cases of mitral regurgitation, when the lesion is not compensated by exactly sufficient hypertrophy of the ventricle, the symptoms may be any of those which we have already considered early in these lectures. It is unnecessary to revert to them.

With regard to *Prognosis* in stenosis and insufficiency respectively, opinions are divided. For my own part, whilst agreeing that in stenosis there are certain special dangers, I am inclined to the belief that on the whole compensation is more certain and more persistent in stenosis.

We come now to consider the case wherein the conditions we have just discussed coexist.

A murmur is heard antecedently to the first sound, and in addition a murmur is heard supplanting, or occurring with, the first sound. The condition is one of combined MITRAL OBSTRUCTION and MITRAL REGURGITATION.

In a very small minority of cases the sound is distinctly double. At the situation of the apex-beat, you hear a murmur with the systole, then a pause,

then a murmur which seems to be diastolic, and then a short pause before the recurrence of the first-sound murmur. You can convince yourself, by carefully timing, that the murmur which appears to be diastolic is really subsequent to the click of the semilunar valves. I am aware that in saying that there may be a pause between a presystolic and a systolic murmur, I am diverging from the teaching of some who have deeply studied the question, but I do not speak without practical experience. I had an opportunity of observing a case in which this was exemplified. At a spot just internal to the apex there were two distinct murmurs separated by pauses. The explanation of the phenomenon appeared to be given by another case under my care, in which the only endocardial murmur was presystolic, but this was separated by a decided pause from the first sound. It was of such character that I called it a diastolic murmur, though I became convinced subsequently that it occurred after the second sound. In this case the post-mortem examination revealed extreme narrowing of the mitral orifice, but in addition the ventricle was, in great measure, filled by vegetations depending from the lower surface of the valve. The latter condition seemed to me to explain the peculiarity of the murmur—the ventricle being already partially filled by the vegetations, the murmur occurred only at the early part of the auricular contraction, ceasing early because the small ventricular space left became quickly replete.

In the great majority of cases the double mitral murmur can be readily resolved into two murmurs, having the distinctive characters which we have already discussed. The systolic element of the com-

pound murmur is widely diffused, but the presystolic is heard only over its usual limited area. In the near neighbourhood of the apex the sound may be distinctly double: you hear the rolling presystolic murmur pass into the prolonged systolic. Or the presystolic may abruptly cease with a sharp first sound from which a soft systolic murmur tails off. In many cases, however, there is no spot where you can hear the two murmurs at one and the same time. This was well described by the late Dr. Salter, who cited a case. In Dr. Salter's words—"At the apex a grinding bruit is heard, *immediately preceding an apparently natural first sound*, and terminated by it. On working back well into the axilla, and not until the axilla is quite reached, a *systolic* murmur begins to reveal itself, increasing in distinctness as you work round to the back, where it is loud and strong. It is audible over the whole of the left side of the back. On returning again to the front, to the region of the apex, it is quite lost, and the presystolic bruit is again heard."* I have no doubt that you will meet with examples verifying this description.

The condition of combined obstruction and insufficiency of the mitral orifice is one that calls for a very grave prognosis.

We may now briefly consider COMBINED MURMURS. We have already discussed double murmurs, that is to say, murmurs generated at different periods of the heart's rhythm at one orifice: by "combined" murmurs I mean those which take their origin from more than one of the orifices. In the hundred cases

* *Lancet*, July 24th, 1869, p. 114.

of heart disease which I have cited, I found such combinations as gave rise to the following diagnoses in the proportions indicated :—

(1.) Mitral regurgitation and aortic obstruction, six cases.

(2.) Mitral regurgitation and aortic regurgitation, five cases.

(3.) Mitral regurgitation, aortic obstruction and regurgitation, five cases.

(4.) Mitral stenosis and aortic regurgitation, one case.

(5.) Mitral regurgitation and tricuspid regurgitation, one case.

None of these combinations require particular comment from the point of view of diagnosis, except the fourth. You will distinguish them by the rules already laid down for the diagnosis of the individual affections. In all cases notice: 1. The positions of maximum intensity of any murmurs heard over the cardiac area. 2. Any differences of pitch and character. 3. The directions in which the sound is conveyed.

There may be considerable difficulty in determining the coexistence of aortic insufficiency and mitral narrowing. A prolonged diastolic murmur may drown the presystolic. In carrying the stethoscope, however, down the left border of the sternum you may probably arrive at a spot where the diastolic murmur ceases to be audible, and then as you approach the apex, a presystolic of different pitch and character may become manifest. The presence of a presystolic thrill at the apex would complete the diagnosis.

I have never met with a case of coexistent mitral

and tricuspid stenosis, but Dr. Hayden, of Dublin, has recorded three such examples. The presence of tricuspid stenosis is to be suspected whenever a presystolic murmur is heard close to the left edge of the sternum. Dr. Hayden "would regard the existence of two centres of presystolic murmur with or without fremitus—viz., at the apex and somewhat to the right of that situation, in conjunction with marked systemic venous engorgement, as evidence of the double lesion of mitral and tricuspid stenosis."*

The diagnosis of complex pathological conditions of the heart sometimes presents very considerable difficulties, and requires great care and repeated examinations. I would venture to give you one or two rules to observe when you meet with a difficult case.

In the first place do not be content to write in your notes, "rhythm of the heart irregular and tumultuous," but let there be order in your record of such irregularity, and system in your treatment of the seeming chaos.

Record all the signs which you have observed previously to those derived from auscultation.

Describe the sounds, normal and abnormal, heard over the situations of each of the orifices.

Note, first, the characters of first sound and second sound at the aortic cartilage. Reduce these to diagrammatic form (see figs. 5, 6, and 7), indicating murmurs where present.

Repeat the process at the pulmonary, tricuspid, and mitral areas successively.

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* "The Diseases of the Heart and of the Aorta," by Thomas Hayden, &c. &c. Dublin: Fannin & Co., 1875, p. 238.

Compare the observations and diagrams only after they have been completed, and then fill in the lines of conduction of normal and abnormal sounds.

Do not unduly hasten to form your conclusions, but obtain all the evidence before you give your verdict.

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